ANSWER, 1 OF 61 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

- AN 1983:240354 BIOSIS
- DN BA75:90354
- TI INFLUENCE OF 6 METHOXY CUMARANON 2-ACETIC-ACID AND ITS DERIVATIVES ON THE PROCESS OF HUMORAL AND CELLULAR IMMUNOGENESIS.
- AU ZABLOCKI B; KIERONSKA D; MARCZYK B; POZALSKA B; LAUK-PUCHALA B
- CS DEP. IMMUNOL., INSTITUTE MICROBIOL., UNIV. LODZ, BANACHA 12/16, 90-237 LODZ.
- SO ARCH IMMUNOL THER EXP, (1981 (RECD 1983)) 29 (6), 805-812. CODEN: AITEAT. ISSN: 0004-069X.
- FS BA; OLD
- LA English
- The influence of 6-metoxy-cumaranon-2-acetic acid and its 4 derivatives ΑB [6-hydroxy-cumaranon-2-acetic acid, 6-hydroxy-cumaranon-2-(N-4carboxyphenyl-)acetamide, 6-hydroxy-cumaranon-2-(N-4carboxymethylphenyl-) acetamide (HKCMF) and 6-hydroxy-cumaranon-2-(N-4carboxy-3-hydroxy-phenyl-) acetamide (HKCHF)] on humoral and cellular immunogenesis was determined in mice. The study of immunosuppressive properties included: control of the level of peripheral blood lymphocytes, estimation of transplantation immunity, GvH [graft vs. host] reaction, PFC [plaque-forming cell] production for SRBCC [sheep erythrocyte] and LPS [lipolysaccharide], and the determintion of the number of cells possessing receptors for antigen, Fc fragment and complement. Blast transformation of lymphocytes stimulated with PHA [phytohemagglutinin] and the cytotoxic effect of sensitized lymphocytes were estimated. Results indicate immunosuppressive activity of acetamides HKCMF and HKCHF. They lowered the level of circulating lymphocytes, the number of cells possessing receptors for complement and they hindered PFC production for SRBC and LPS. Acetamide HKCHF weakened the cytotoxic activity of lymphocytes sensitized to alloantigens.
- The influence of 6-metoxy-cumaranon-2-acetic acid and its 4 derivatives [6-hydroxy-cumaranon-2-acetic acid, 6-hydroxy-cumaranon-2-(N-4-carboxymethylphenyl-) acetamide, 6-hydroxy-cumaranon-2-(N-4-carboxy-3-hydroxy-phenyl-) acetamide (HKCMF) and 6-hydroxy-cumaranon-2-(N-4-carboxy-3-hydroxy-phenyl-) acetamide (HKCHF)] on humoral and cellular immunogenesis was determined in mice. The study of immunosuppressive.
- IT Miscellaneous Descriptors
 - MOUSE SHEEP LYMPHOCYTO TOXICITY PLAQUE FORMING CELL ERYTHROCYTE 6
 HYDROXY CUMARANON-2-ACETIC-ACID 6 HYDROXY CUMARANON 2-N-4
 CARBOXYPHENYL ACETAMIDE 6 HYDROXY CUMARANON-2 N-4
 CARBOXYMETHYLPHENYL ACETAMIDE 6 HYDROXY CUMARANON 2-N-4
 CARBOXY-3-HYDROXYPHENYL ACETAMIDE IMMUNOLOGIC-DRUG IMMUNO SUPPRESSANT
 PHYTO HEM AGGLUTININ FC RECEPTOR COMPLEMENT GRAFT
 VS. HOST REACTION
- L8 ANSWER 2 OF 61 BIOTECHNO COPYRIGHT 2002 Elsevier Science B.V.
- AN 1991:21339261 BIOTECHNO
- TI Immunologically activated chloride channels involved in degranulation of rat mucosal mast cells
- AU Romanin C.; Reinsprecht M.; Pecht I.; Schindler H.
- CS Institute for Biophysics, University of Linz, A-4040 Linz, Austria.
- SO EMBO Journal, (1991), 10/12 (3603-3608)
 - CODEN: EMJODG ISSN: 0261-4189
- DT Journal; Article
- CY United Kingdom
- LA English
- SL English
- Crosslinking of type I Fc(.epsilon.) receptors (Fc(.epsilon.)RI) on the surface of basophils or mast cells initiates a cascade of processes leading to the secretion of inflammatory mediators. We report here a correlation between mediator secretion and the activation of Cl.sup.-channels in rat mucosal-type mast cells (line RBL-2H3). Stimulation of RBL cells by either IgE and antigen or by a monoclonal antibody specific for the Fc(.epsilon.)RI, resulted in the activation of Cl.sup.-ion channels as detected by the patch-clamp technique. Channel activation occurred slowly, within minutes after stimulation. The channel has a

slope conductance of 32 pS at potentials between 0 and -100 mV, and an increasing open-state probability with increasing depolarization. Activation of apparently the same Cl.sup. - channels could be mimicked without stimulation by isolating inside-out membrane patches in tyrode solution. Parallel inhibition of both Cl.sup.- channel activity and mediator secretion, as monitored by serotonin release, was observed by two compounds, the Cl.sup.- channel blocker 5-nitro-2-(3phenylpropylamino) benzoic acid (NPPB) and the anti-allergic drug cromolyn. NPPB inhibited both the antigen-induced Cl.sup. - current and the serotonin release, where half-maximal inhibition occurred at similar doses, at 52 .mu.M and 77 .mu.M, respectively. The drug cromolyn, recently found to inhibit immunologically induced mediator secretion from RBL cells upon intracellular application, also blocks Cl.sup. - channels (IC.sub.5.sub.0 = 15 .mu.M) when applied to the cytoplasmic side of an inside-out membrane patch. The observed Cl.sup.channel activation upon immunological stimulation and the parallel inhibition of channel current and of serotonin release suggests a functional role for this Cl.sup. - channel in mediator secretion from the mast cells studied.

- channel activity and mediator secretion, as monitored by serotonin AB. release, was observed by two compounds, the Cl.sup. - channel blocker 5-nitro-2-(3-phenylpropylamino) benzoic acid (NPPB) and the anti-allergic drug cromolyn. NPPB inhibited both the antigen-induced Cl.sup. - current and the serotonin release, where half-maximal inhibition.
- *chloride channel; *immunostimulation; *serotonin release; Fc CT receptor; 5 nitro 2 (3 phenylpropylamino)benzoic acid; cromoglycate disodium; monoclonal antibody; animal cell; article; cross linking; degranulation; mast cell degranulation; mediator; mucosa cell; nonhuman; patch clamp; priority.
- RN(5 nitro 2 (3 phenylpropylamino)benzoic acid) 107254-86-4; (cromoglycate disodium) 15826-37-6, 16110-51-3, 93356-79-7, 93356-84-4
- ANSWER 3 OF 61 CAPLUS COPYRIGHT 2002 ACS L8
- 1995:644214 CAPLUS AN
- DN 123:54051
- The effect of a naphthalene derivative, TEI-6472, on histamine release and TI tyrosine phosphorylation in rat basophilic leukemia cells
- Miyamoto, Hisashi; Matsui, Katsuhiko; Arai, Toshihiko ΑIJ
- CS Dep. Microbiology, Meiji Coll. Pharmacy, Tokyo, 154, Japan
- SO Int. J. Immunopharmacol. (1995), 17(5), 433-41 CODEN: IJIMDS; ISSN: 0192-0561
- DΤ Journal
- LΑ English
- AB. It is well known that rat basophilic leukemia cells (RBL-2H3) express high-affinity IgE receptors (Fc.epsilon.RI) and that the aggregation of these receptors causes the release of chem. mediators. When RBL-2H3 cells are sensitized with IgE and subsequently stimulated by an antigen, histamine release and the tyrosine phosphorylation of several proteins are obsd. Here, the authors examd. the effects of a synthetic naphthalene deriv., (7E)-N-(2-carboxyphenyl)-8-(2-naphthyl)-5,6-trans-5,6methano-7-octenamide (TEI-6472), on the Fc.epsilon.RI-mediated histamine release from RBL-2H3 cells. Preincubation for 10 min with 100 .mu.M TEI-6472 caused inhibition of Fc.epsilon.RI-mediated histamine release from RBL-2H3 cells. Furthermore, Western blotting anal. using anti-phosphotyrosine antibody showed that Fc.epsilon.RI-mediated tyrosine phosphorylation of 78 and 92 kDa proteins in RBL-2H3 cells was also inhibited. Tyrosine phosphorylation of these 78 and 92 kDa proteins was not induced by direct activation of protein kinase C (PKC) by phorbol-12-myristate-13-acetate (PMA) and the calcium ionophore A23187. However, the inhibition of histamine release from TEI-6472-treated RBL-2H3 cells was restored by direct activation of PKC. Thus, tyrosine phosphorylation of the 78 and 92 kDa proteins in RBL-2H3 cells is involved in a signal transduction system for histamine secretion and the phosphorylation may occur upstream of PKC activation. AB
 - It is well known that rat basophilic leukemia cells (RBL-2H3) express

high-affinity IgE receptors (Fc.epsilon.RI) and that the aggregation of these receptors causes the release of chem. mediators. When RBL-2H3 cells are sensitized with IgE and subsequently stimulated by an antigen, histamine release and the tyrosine phosphorylation of several proteins are obsd. Here, the authors examd. the effects of a synthetic naphthalene deriv., (7E)-N-(2-carboxyphenyl)-8-(2-naphthyl)-5,6-trans-5,6methano-7-octenamide (TEI-6472), on the Fc.epsilon.RI-mediated histamine release from RBL-2H3 cells. Preincubation for 10 min with 100 .mu.M TEI-6472 caused inhibition of Fc.epsilon.RI-mediated histamine release from RBL-2H3 cells. Furthermore, Western blotting anal. using anti-phosphotyrosine antibody showed that Fc.epsilon.RI-mediated tyrosine phosphorylation of 78 and 92 kDa proteins in RBL-2H3 cells was also inhibited. Tyrosine phosphorylation of these 78 and 92 kDa proteins was not induced by direct activation of protein kinase C (PKC) by phorbol-12-myristate-13-acetate (PMA) and the calcium ionophore A23187. However, the inhibition of histamine release from TEI-6472-treated RBL-2H3 cells was restored by direct activation of PKC. Thus, tyrosine phosphorylation of the 78 and 92 kDa proteins in RBL-2H3 cells is involved in a signal transduction system for histamine secretion and the phosphorylation may occur upstream of PKC activation.

ΙT Immunoglobulin receptors

Receptors

RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)

(Fc.epsilon.RI (IgE fragment Fc receptor I), tyrosine phosphorylation upstream of protein kinase C activation is involved in Fc.epsilon.RI-mediated signaling for histamine secretion in basophils)

- L8 ANSWER 4 OF 61 CAPLUS COPYRIGHT 2002 ACS
- AN 1992:5109 CAPLUS
- DN 116:5109
- TIImmunologically activated chloride channels involved in degranulation of rat mucosal mast cells
- ΑU Romanin, Christoph; Reinsprecht, Martin; Pecht, Israel; Schindler, Hansgeorg
- Inst. Biophys., Univ. Linz, Linz, A-4040, Austria CS
- SO EMBO J. (1991), 10(12), 3603-8 CODEN: EMJODG; ISSN: 0261-4189
- 'nΨ Journal
- LΑ English
- AΒ Crosslinking of type I Fc.epsilon. receptors (Fc.epsilon.RI) on the surface of basophils or mast cells initiates a cascade of processes leading to the secretion of inflammatory mediators. The authors report here a correlation between mediator secretion and the activation of C1channels in rat mucosal-type mast cells (line RBL-2H3). Stimulation of RBL cells by either IgE and antigen or by a monoclonal antibody specific for the Fc.epsilon.RI, resulted in the activation of Cl- ion channels as detected by the patch-clamp technique. Channel has a slope conductance of 32 pS at potentials between 0 and -100 mV, and an increasing open-state probability with increasing depolarization. Activation of apparently the same Cl- channels could be mimicked without stimulation by isolating inside-out membrane patches in Tyrode soln. Parallel inhibition of both Cl- channel activity and mediator secretion, as monitored by serotonin release, was obsd. by two compds., the Cl- channel blocker 5-nitro-2-(3-phenylpropylamino) benzoic acid (NPPB) and the anti-allergic drug cromolyn. NPPB inhibited both the antigen-induced Cl- current and the serotonin release, where half-maximal inhibition occurred at similar doses, at 52 .mu.M and 77 .mu.M, resp. drug cromolyn, recently found to inhibit immunol. induced mediator secretion from RBL cells upon intracellular application, also blocks Clchannels (IC50 = 15 .mu.M) when applied to the cytoplasmic side of an inside-out membrane patch. The obsd. Cl- channel activation upon immunol. stimulation and the parallel inhibition of channel current and of serotonin release suggests a functional role for this C1- channel in mediator secretion from the mast cells studied. AB
 - Crosslinking of type I Fc.epsilon. receptors (Fc.epsilon.RI) on the

surface of basophils or mast cells initiates a cascade of processes leading to the secretion of inflammatory mediators. The authors report here a correlation between mediator secretion and the activation of Clchannels in rat mucosal-type mast cells (line RBL-2H3). Stimulation of RBL cells by either IgE and antigen or by a monoclonal antibody specific for the Fc.epsilon.RI, resulted in the activation of Cl- ion channels as detected by the patch-clamp technique. Channel has a slope conductance of 32 pS at potentials between 0 and -100 mV, and an increasing open-state probability with increasing depolarization. Activation of apparently the same Cl- channels could be mimicked without stimulation by isolating inside-out membrane patches in Tyrode soln. Parallel inhibition of both Cl- channel activity and mediator secretion, as monitored by serotonin release, was obsd. by two compds., the Cl- channel blocker 5-nitro-2-(3-phenylpropylamino) benzoic acid (NPPB) and the anti-allergic drug cromolyn. NPPB inhibited both the antigen-induced Cl- current and the serotonin release, where half-maximal inhibition occurred at similar doses, at 52 .mu.M and 77 .mu.M, resp. drug cromolyn, recently found to inhibit immunol. induced mediator secretion from RBL cells upon intracellular application, also blocks C1channels (IC50 = 15 .mu.M) when applied to the cytoplasmic side of an inside-out membrane patch. The obsd. Cl- channel activation upon immunol. stimulation and the parallel inhibition of channel current and of serotonin release suggests a functional role for this Cl- channel in mediator secretion from the mast cells studied.

ΙT Receptors

(Fc.epsilon.RI (IgE fragment Fc receptor I), chloride channel activation coupled to, in degranulation of mucosal mast cell) .

ANSWER 5 OF 61 DRUGU COPYRIGHT 2002 THOMSON DERWENT L8

1994-28905 DRUGU ΑN

Synthetic ligand to the retinoid X receptor (RXR) synergizes with either ΤI all-trans (T-) or 9-Cis (9-C-) retinoic acid (RA) to induce differentiation of myeloid leukemic cells.

Kerner B; Dawson M I; Faucett C; Koeffler H P ΑU

CS Univ. California

Los Angeles, Menlo Park, California, United States LO SO

Proc.Am. Assoc. Cancer Res. (35, 85 Meet., 274, 1994)

Department of Medicine, Cedars-Sinai Medical Center, UCLA, CA 90048, ΑV U.S.A.

LA English

DT Journal

FA AB; LA; CT

FS Literature

AN 1994-28905 DRUGU

The synthetic ligand (4-(2-methyl-1-(5,6,7,8, -tetrahydro-5,5,8,8, AB. tetramethyl 2-naphthalenyl) propen-1-yl)benzoic acid, (SR-11217) to the retinoid X receptor (RXR) synergized with either all trans or 9-cis retinoic acid (RA) to induce differentiation of myeloid leukemic cells. SRI-11217 alone, had almost no activity. SRI-11217 had no effect on expression of the low affinity Fc receptor for IqE (CD23). Combinations of SRI-11217 with either T-RA or 9-C-RA synergistically increased expression of CD23. These results show that a ligand to the RXR synergize with a ligand to retinoic acid receptor (RAR). Furthermore, the data suggest that amplification of the hormone response to T-RA is possible with ligands specific for RXR. (conference abstract).

SRI-11217 alone, had almost no activity. There was a synergistic effect ABEX of low concentrations of either 9-C-RA or T-RA with SRI-11217, as measured by inhibition of clonal proliferation and induction of differentiation of HL-60 cells. The expression of the low affinity Fc receptor for IqE (CD23), which is up-regulated during RA-induced myeloid differentiation was examined. Treatment of HL-60 cells with either T-RA or 9-C-RA (10 nM) for 5 days moderately increased the expression of CD23 antigen. In contrast, SRI-11217 (1 uM-10 nM) had no effect on expression of this receptor. Combination of SRI-11217 (1 uM) with either T-RA or 9-C-RA synergistically increased

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The synthetic ligand (4-(2-methyl-1-(5,6,7,8, -tetrahydro-5,5,8,8,
AΒ
      tetramethyl 2-naphthalenyl) propen-1-yl)benzoic acid,
      (SR-11217) to the retinoid X receptor (RXR) synergized with either all
      trans or 9-cis retinoic acid (RA) to induce differentiation.
      myeloid leukemic cells. SRI-11217 alone, had almost no activity.
      SRI-11217 had no effect on expression of the low affinity {\bf Fc}
      receptor for IgE (CD23). Combinations of SRI-11217 with either
      T-RA or 9-C-RA synergistically increased expression of CD23. These
      results show that.
      . . as measured by inhibition of clonal proliferation and induction of
ABEX.
      differentiation of HL-60 cells. The expression of the low affinity
      Fc receptor for IgE (CD23), which is up-regulated
      during RA-induced myeloid differentiation was examined. Treatment of
      HL-60 cells with either T-RA or.
         MYELOID *FT; IN-VITRO *FT; TUMOR-CELL *FT; LEUKEMIA *FT; COMB. *FT;
CT
         FC-RECEPTOR *FT; EXPRESSION *FT; CD23 *FT;
         CYTOSTATIC *FT; TISSUE-CULTURE *FT
       MYELOID *FT; IN-VITRO *FT; TUMOR-CELL *FT; LEUKEMIA *FT; COMB. *FT;
        FC-RECEPTOR *FT; EXPRESSION *FT; CD23 *FT;
         CYTOSTATIC *FT; TISSUE-CULTURE *FT
     ANSWER 6 OF 61 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
r_8
AN .
     2001225012 EMBASE
TΙ
     Cytotoxicity testing of wound-dressing materials.
ΑU
     Sahlin H.; Nygren H.
     H. Sahlin, Applied Cell Biology, Department of Anatomy, Goteborg
CS
     University, P.O. Box 420, 405 30 Gothenburg, Sweden
     ATLA Alternatives to Laboratory Animals, (2001) 29/3 (269-275).
SO
     Refs: 13
     ISSN: 0261-1929 CODEN: AALADQ
CY
     United Kingdom
DT
     Journal; Conference Article
FS
     052
             Toxicology
LА
     English
SL
     English
AΒ
     A method was developed for testing the cytotoxicity of various
     bandage-like wound dressings and gel wound dressings. In this method, the
     ability of human polymorphonuclear neutrophils (PMNs) to initiate a
     respiratory burst after exposure to the various wound dressings is used as
     a marker of cytotoxicity. Luminol-amplified chemiluminescence stimulated
     with opsonised zymosan or phorbol 12-myristate 13-acetate (PMA) is used to
     measure the degree of activation of the respiratory burst, i.e. the NADPH
     oxidase activity, after exposure to wound dressings. Opsonised zymosan
     (material from yeast cell walls) is a phagocytic stimulus that activates
     the NADPH oxidase by binding to Fe-receptors and complement receptors, and
     functions as an artificial bacterium, whereas PMA activates the NADPH
     oxidase by direct activation of protein kinase C. NADPH oxidase activity
     was inhibited by several wound dressings. The down-regulation of the
     respiratory burst is detrimental to the bactericial effect of PMNs, and
     can be used as a marker for the cytotoxicity of wound-dressing materials.
     Medical Descriptors:
                           . paper
     *immunotoxicity: .
     priority journal
     *gelling agent: TO, drug toxicity
     acemannan: TO, drug toxicity
     zymosan
     reduced nicotinamide adenine dinucleotide dehydrogenase: EC, endogenous
     compound
     phorbol 13 acetate 12 myristate
       Fc receptor: EC, endogenous compound
     complement receptor: EC, endogenous compound
     carbomer: TO, drug toxicity
     povidone: TO, drug toxicity
     alginic acid: TO, drug toxicity
     carboxymethylcellulose: TO, drug toxicity
     guar gum: TO, drug toxicity
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expression of CD23. (KJ)

pectin: TO, drug toxicity xanthan: TO, drug toxicity collagen: TO, drug toxicity sorbate potassium: TO, drug toxicity benzoic acid: TO, drug toxicity sodium metabisulfite: TO, drug toxicity propylene glycol: TO, drug toxicity methyl paraben: TO, drug toxicity germall 115: TO, drug toxicity propyl. 29894-36-8, 9005-32-7, 9005-38-3; (carboxymethylcellulose) 8050-38-2, 9000-11-7, 9004-32-4, 9050-04-8; (guar gum) 9000-30-0; (pectin) 9000-69-5; (xanthan) 11138-66-2; (collagen) 9007-34-5; (sorbate potassium) 24634-61-5; (benzoic acid) 532-32-1, 582-25-2, 65-85-0, 766-76-7; (sodium metabisulfite) 7681-57-4, 7757-74-6; (propylene glycol) 57-55-6; (methyl paraben) 99-76-3; (germall 115) 39236-46-9; (propyl paraben) 94-13-3 ANSWER 7 OF 61 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V. 1999108984 EMBASE 4-Trifluoromethyl derivatives of salicylate, triflusal and its main metabolite 2-hydroxy-4-trifluoromethylbenzoic acid, are potent inhibitors of nuclear factor .kappa.B activation. Bayon Y.; Alonso A.; Crespo M.S. M.S. Crespo, Inst. Biologia y Genetica Molecular, CSIC, Facultad de Medicina, 47005-Valladolid, Spain. mscres@ibgm.uva.es British Journal of Pharmacology, (1999) 126/6 (1359-1366). Refs: 40 ISSN: 0007-1188 CODEN: BJPCBM United Kingdom Journal; Article Clinical Biochemistry 029 030 Pharmacology 037 Drug Literature Index English English 1. The effect of two derivatives of salicylate, 2-hydroxy-4trifluoromethylbenzoic acid (HTB) and 2-acetoxy-4-trifluoromethylbenzoic acid (triflusal), on the activation of NF-.kappa.B elicited by tumour necrosis factor-.alpha. (TNF-.alpha.) on human umbilical vein endothelial cells (HUVEC) was tested. 2. The expression of the mRNA of vascular cell adhesion molecule-1 (VCAM-1) was studied as an example of a gene the expression of which is regulated by NF-.kappa.B. To extend these findings to other systems, the induction of nitric oxide synthase in rat adherent peritoneal macrophages was studied. 3. Both HTB and triflusal were more potent than aspirin or salicylate as inhibitors of the nuclear translocation of NF-.kappa.B. The calculation of the IC50 values showed .simeq. 2 mM for HTB, 4 mM for aspirin and > 4 mM for salicylate. 4. Comparison of the potency of these compounds on VCAM-1 mRNA expression showed complete inhibition by both triflusal and HTB at a concentration of 4 mM whereas aspirin and salicylate produced only 36-43% inhibition at the same concentration. 5. Inhibition of NF-.kappa.B.activation was also observed in rat peritoneal macrophages stimulated via their receptors for the Fc portion of the antibody molecule with IgG/ovalbumin immune complexes. This was accompanied by a dose-dependent inhibition of nitrite production by the L-arginine pathway via iNOS. IC50 values for this effect were 1.13 .+-. 0.12 mM (triflusal), 1.84 .+-. 0.34 (HTB), 6.08 .+-. 1.53 mM (aspirin) and 9.16 .+-. 1.9 mM \setminus (salicylate). 6. These data indicate that the incorporation of a 4-trifluoromethyl group to the salicylate molecule strongly enhances its inhibitory effect on NF-.kappa.B activation, VCAM-1 mRNA expression and iNOS induction, irrespective of the presence of the acetyl moiety involved in the inhibition of cyclo-oxygenase. Medical Descriptors: *transcription . . DV, drug development *salicylic acid derivative: PD, pharmacology

RN.

L8°

TI

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AΒ

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*triflusal: CM, drug comparison
*triflusal: DV, drug development
*triflusal: PD, pharmacology
*immunoglobulin enhancer binding protein: EC, endogenous compound
 benzoic acid derivative: CM, drug comparison
 benzoic acid derivative: DV, drug development
 benzoic acid derivative: PD, pharmacology
tumor necrosis factor alpha
messenger RNA: EC, endogenous compound
vascular cell adhesion moxecule 1: EC, endogenous compound
nitric oxide synthase: EC, endogenous compound
salicylic acid: CM, drug comparison
acetylsalicylic acid: CM, drug comparison
  Fc receptor: EC/ endogenous compound
nitrite: EC, endogenous compound
ANSWER 8 OF 61 'EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
95173813 EMBASE
1995173813
The effect of a naphthalene derivative, TEI-6472, on histamine release and
tyrosine phosphorylation in rat basophilic leukemia cells.
Miyamoto H.; Matsui K.; Arai T.
Department of Microbiology, Meiji College of Pharmacy, 1-35-23
Nozawa, Setagya-ku, Tokyo 154, Japan
International Journal of Immunopharmacology, (1995) 17/5 (433-441).
ISSN: 0192-0561 CODEN: IJIMDS
United Kingdom
Journal; Article
016
025
        Hematology
026
        Immunology, Serology and Transplantation
        Pharmacology
030
037
        Drug Literature Index
English
English
It is well known that rat basophilic leukemia cells (RBL-2H3) express
high-affinity IgE receptors (Fc.epsilon.RIU) and that the aggregation of
these receptors causes the release of chemical mediators. When RBL-2H3
cells are sensitized with IgE antibody and subsequently stimulated by an
antigen, significant histamine release and the tyrosine phosphorylation of
several proteins are observed. In this study, we examined the effects of a
synthetic naphthalene derivative, (7E)-N-(2-carboxyphenyl
)-8-(2-naphthyl)-5,6-trans-5,6-methano-7-octenamide (TEI-6472), on the
Fc.epsilon.RI-mediated histamine release From RBL-2H3 cells. Preincubation
for 10 min with 100 .mu.M TEI-6472 caused significant inhibition of
Fc.epsilon.RI-mediated histamine release from RBL-2H3 cells. Furthermore,
Western blotting analysis using anti-phosphotyrosine antibody showed that
Fc.epsilon.RI-mediated tyrosine phosphorylation of 78 and 92 kDa proteins
in RBL-2H3 cells was also significantly inhibited. Tyrosine
phosphorylation of these 78 and 92 kDa proteins was not induced by direct
activation of protein kinase C (PKC) by phorbol-12-myristate-13-acetate
(PMA) and the calcium ionophore A23187. However, the inhibition of
histamine release from TEI-6472-treated RBL-2H3 cells was restored by
direct activation of PKC. Taken together, these results suggest that
tyrosine phosphorylation of the 78 and 92 kDa proteins in RBL-2H3 cells is
involved in a signal transduction system for histamine secretion, and that
these tyrosine phosphorylations may occur upstream of PKC activation.
  . . the tyrosine phosphorylation of several proteins are observed. In
this study, we examined the effects of a synthetic naphthalene derivative,
(7E)-N-(2-carboxyphenyl)-8-(2-naphthyl)-5,6-trans-5,6-methano-7-
octenamide (TEI-6472), on the Fc.epsilon.RI-mediated histamine release
From RBL-2H3 cells. Preincubation for 10 min with 100 .mu.M TEI-6472
caused significant inhibition. .
Medical Descriptors:
*histamine release
*mast cell leukemia
*protein phosphorylation
```

L8 AN

·DN

ΤI

ΑU

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DT

FS

LA SL

AB

AB

CT

*signal transduction animal cell article nonhuman priority journal rat

Fc receptor

*histamine: EC, endogenous compound *immunoglobulin e: EC, endogenous compound *immunomodulating agent: PD, pharmacology *naphthalene derivative: PD, pharmacology *phorbol ester *protein kinase c: EC, endogenous compound

L8 ANSWER 9 OF 61 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

AN 91339955 EMBASE

DN 1991339955

TI Immunologically activated chloride channels involved in degranulation of rat mucosal mast cells.

AU Romanin C.; Reinsprecht M.; Pecht I.; Schindler H.

CS Institute for Biophysics, University of Linz, A-4040 Linz, Austria

EMBO Journal, (1991) 10/12 (3603-3608).

ISSN: 0261-4189 CODEN: EMJODG

CY United Kingdom

DT Journal; Article

FS 026 Immunology, Serology and Transplantation

LA English

SO

SL English

AΒ Crosslinking of type I Fc(.epsilon.) receptors (Fc(.epsilon.)RI) on the surface of basophils or mast cells initiates a cascade of processes leading to the secretion of inflammatory mediators. We report here a correlation between mediator secretion and the activation of Cl- channels in rat mucosal-type mast cells (line RBL-2H3). Stimulation of RBL cells by either IgE and antigen or by a monoclonal antibody specific for the Fc(.epsilon.)RI, resulted in the activation of Cl- ion channels as detected by the patch-clamp technique. Channel activation occurred slowly, within minutes after stimulation. The channel has a slope conductance of $32~\mathrm{pS}$ at potentials between 0 and $-100~\mathrm{mV}$, and an increasing open-state probability with increasing depolarization. Activation of apparently the same Cl- channels could be mimicked without stimulation by isolating inside-out membrane patches in tyrode solution. Parallel inhibition of both Cl- channel activity and mediator secretion, as monitored by serotonin release, was observed by two compounds, the Cl- channel blocker 5-nitro-2-(3-phenylpropylamino) benzoic acid (NPPB) and the anti-allergic drug cromolyn. NPPB inhibited both the antigen-induced Cl- current and the serotonin release, where half-maximal inhibition occurred at similar doses, at 52 .mu.M and 77 .mu.M, respectively. The drug cromolyn, recently found to inhibit immunologically induced mediator secretion from RBL cells upon intracellular application, also blocks Cl- channels (IC50 = 15 .mu.M) when applied to the cytoplasmic side of an inside-out membrane patch. The observed Cl- channel activation. upon immunological stimulation and the parallel inhibition of channel current and of serotonin release suggests a functional role for this Clchannel in mediator secretion from the mast cells studied.

AB . . . channel activity and mediator secretion, as monitored by serotonin release, was observed by two compounds, the Cl- channel blocker 5-nitro-2-(3-phenylpropylamino) benzoic acid (NPPB) and the anti-allergic drug cromolyn. NPPB inhibited both the antigen-induced Cl- current and the serotonin release, where half-maximal inhibition. . .

CT Medical Descriptors:

*chloride channel

*immunostimulation

*serotonin release

animal cell

article

```
cross linking
degranulation
mast cell degranulation
mediator
mucosa cell
nonhuman
patch clamp
priority journal
secretion
stimulation
  Fc receptor
  5 nitro 2 (3 phenylpropylamino) benzoic acid
cromoglycate disodium
monoclonal antibody
(5 nitro 2 (3 phenylpropylamino)benzoic acid)
107254-86-4; (cromoglycate disodium) 15826-37-6, 16110-51-3, 93356-79-7,
93356-84-4
                                  rgulugasan i kambar i kukasasa ki kasarau.
ANSWER 10 OF 61 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
77121160 EMBASE
1977121160
Receptors for antibody opsonic adherence on the eosinophils of guinea
Butterworth A.E.; Coombs R.R.A.; Gurner B.W.; Wilson A.B.
Div. Immunol., Dept. Pathol., Univ. Cambridge, United Kingdom
International Archives of Allergy and Applied Immunology, (1976) 51/3
(368-377).
CODEN: IAAAAM
Journal
026
        Immunology, Serology and Transplantation
025
        Hematology
English
Eosinophils have recently been implicated in antibody dependent cell
mediated damage to schistosomula. Because of this, eosinophils of the
guinea pig have been examined for surface receptors capable of giving
antibody opsonic adherence; a rosetting reaction has been used. The
eosinophils were shown to possess Fc receptors for homologous
immunoglobulin. No selective difference between IgG1 and IgG2 was
observed. In marked contrast to macrophages, guinea pig eosinophils failed
to show opsonic adherence to red cells sensitized to a comparable degree
with rabbit antibody. With red cell antibodies made in the pig, however,
the reciprocal situation held, namely opsonic adherence was stronger with
eosinophils than with macrophages.
Medical Descriptors:
  *4 (1 imidazolylmethyl)benzoic acid
*cytolysis
*eosinophil
*humoral immunity
in vitro study
theoretical study
guinea pig
  *Fc receptor
ANSWER 11 OF 61
                    MEDLINE
89008870
             MEDLINE
89008870
           PubMed ID: 3049672
Studies on the molecular mechanisms of human Fc receptor
-mediated phagocytosis. Amplification of ingestion is dependent on the
```

AU Gresham H D; McGarr J A; Shackelford P G; Brown E J
CS Department of Medicine, Washington University School of Medicine, St.
Louis, Missouri 63110.
NC AI-19350 (NIAID)

generation of reactive oxygen metabolites and is deficient in

polymorphonuclear leukocytes from patients with chronic granulomatous

NC AI-19350 (NIAID) AI-23790 (NIAID)

RN

L8 AN

DN

TI

AU CS

SO

DΤ

FS

LA

AΒ

CT

L8

ΑN

DN

TТ

GM-38330 (NIGMS) JOURNAL OF CLINICAL INVESTIGATION, (1988 Oct) 82 (4) 1192-201. Journal code: HS7; 7802877. ISSN: 0021-9738. CY · United States Journal; Article; (JOURNAL ARTICLE) DΨ ĿА English Abridged Index Medicus Journals; Priority Journals FS EM198811 Entered STN: 19900308 ED Last Updated on STN: 19990129 Entered Medline: 19881115 Human PMN and monocytes both possess a mechanism for amplifying Fc AB receptor-mediated phagocytic function, which is dependent on activation of the respiratory burst. The pathway for augmentation of phagocytosis requires superoxide anion, hydrogen peroxide, and lactoferrin and is independent of the hydrogen peroxide-MPO-halide system. In neither cell type is this mechanism induced upon exposure to the opsonized target. PMN require an additional signal for stimulation of the respiratory burst; this is not true of monocytes. On the other hand, monocytes require an exogenous source of lactoferrin in order to activate this pathway for enhanced ingestion. The dependence of this pathway for both PMN and monocytes on superoxide anion, hydrogen peroxide, and cell-bound lactoferrin is consistent with a role for locally generated reactive oxygen metabolites, possibly hydroxyl radicals, in phagocytosis amplification. Patients with chronic granulomatous disease, who are genetically deficient in the ability to activate the respiratory burst, are unable to amplify Fc receptor-mediated phagocytosis. Thus, these patients may have a previously unrecognized defect in the recruitment of phagocytic function at inflammatory sites. ΤI Studies on the molecular mechanisms of human Fc receptor -mediated phagocytosis. Amplification of ingestion is dependent on the generation of reactive oxygen metabolites and is deficient in polymorphonuclear leukocytes from. . . . Human PMN and monocytes both possess a mechanism for amplifying Fc AΒ receptor-mediated phagocytic function, which is dependent on activation of the respiratory burst. The pathway for augmentation of phagocytosis requires superoxide anion,. . . with chronic granulomatous disease, who are genetically deficient in the ability to activate the respiratory burst, are unable to amplify Fc receptor -mediated phagocytosis. Thus, these patients may have a previously unrecognized defect in the recruitment of phagocytic function at inflammatory sites. . Human; Male; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S. CT Amphotericin B: AI, antagonists & inhibitors Amphotericin B: PD, pharmacology Benzoates Benzoic Acid Biological Factors: AI, antagonists & inhibitors Biological Factors: PD, pharmacology Catalase Cytokines Free Radicals *Granulomatous Disease, Chronic: BL,. 1397-89-3 (Amphotericin B); 37558-16-0 (Phorbol 12,13-Dibutyrate); RN65-85-0 (Benzoic Acid); 9010-72-4 (Zymosan) ANSWER 12 OF 61 TOXCENTER COPYRIGHT 2002 ACS L8 1989:4894 TOXCENTER AN 89008870 PubMed ID: 3049672 DN Studies on the molecular mechanisms of human Fc receptor TΙ -mediated phagocytosis. Amplification of ingestion is dependent on the generation of reactive oxygen metabolites and is deficient in

AU Gresham H D; McGarr J A; Shackelford P G; Brown E J
CS Department of Medicine, Washington University School of Medicine, St.
Louis, Missouri 63110

polymorphonuclear leukocytes from patients with chronic granulomatous

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NC
     AI-19350 (NIAID)
        AI-23790 (NIAID)
        GM-38330 (NIGMS)
        JOURNAL OF CLINICAL INVESTIGATION, (1988 Oct) 82 (4) 1192-201.
SO
        Journal Code: HS7; 7802877. ISSN: 0021-9738.
CY
        United States
DΤ
        Journal; Article; (JOURNAL ARTICLE)
FS
        MEDLINE
        MEDLINE 89008870
OS
LA
        English
ED
        Entered STN: 20011116
        Last Updated on STN: 20011116
        1989:4894 TOXCENTER
AN
        Human PMN and monocytes both possess a mechanism for amplifying Fc
AΒ
        receptor-mediated phagocytic function, which is dependent on
        activation of the respiratory burst. The pathway for augmentation of
        phagocytosis requires superoxide anion, hydrogen peroxide, and lactoferrin
        and is independent of the hydrogen peroxide-MPO-halide system. In neither
        cell type is this mechanism induced upon exposure to the opsonized target.
        PMN require an additional signal for stimulation of the respiratory burst;
        this is not true of monocytes. On the other hand, monocytes require an
        exogenous source of lactoferrin in order to activate this pathway for
        enhanced ingestion. The dependence of this pathway for both PMN and
        monocytes on superoxide anion, hydrogen peroxide, and cell-bound
        lactoferrin is consistent with a role for locally generated reactive
        oxygen metabolites, possibly hydroxyl radicals, in phagocytosis
        amplification. Patients with chronic granulomatous disease, who are
        genetically deficient in the ability to activate the respiratory burst,
        are unable to amplify Fc receptor-mediated
        phagocytosis. Thus, these patients may have a previously unrecognized
        defect in the recruitment of phagocytic function at inflammatory sites.
TI
        Studies on the molecular mechanisms of human Fc receptor
        -mediated phagocytosis. Amplification of ingestion is dependent on the
        generation of reactive oxygen metabolites and is deficient in
        polymorphonuclear leukocytes from.
                                                                    . .
        Human PMN and monocytes both possess a mechanism for amplifying Fc
ÀΒ
        receptor-mediated phagocytic function, which is dependent on
        activation of the respiratory burst. The pathway for augmentation of
        phagocytosis requires superoxide anion,. . . with chronic granulomatous
        disease, who are genetically deficient in the ability to activate the
        respiratory burst, are unable to amplify Fc receptor
        -mediated phagocytosis. Thus, these patients may have a previously
        unrecognized defect in the recruitment of phagocytic function at
        inflammatory sites.
                 . Human; Male; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
CT
          Amphotericin B: AI, antagonists & inhibitors
          Amphotericin B: PD, pharmacology
          Benzoates
             Benzoic Acid
          Biological Factors: AI, antagonists & inhibitors
          Biological Factors: PD, pharmacology
          Catalase
                           the control of the co
          Cytokines
          Free Radicals
        *Granulomatous Disease, Chronic: BL, blood
          Granulomatous.
RN
        1397-89-3 (Amphotericin B)
        37558-16-0 (Phorbol 12,13-Dibutyrate)
        65-85-0 (Benzoic Acid)
        9010-72-4 (Zymosan)
L8
        ANSWER 13 OF 61 TOXCENTER COPYRIGHT 2002 ACS
        1983:75994 TOXCENTER
ΑN
        Copyright 2002 BIOSIS
CP
        BA75:90354
DN
        INFLUENCE OF 6 METHOXY CUMARANON 2-ACETIC-ACID AND ITS DERIVATIVES ON THE
TI
```

PROCESS OF HUMORAL AND CELLULAR IMMUNOGENESIS

```
ZABLOCKI B; KIERONSKA D; MARCZYK B; POZALSKA B; LAUK-PUCHALA B
ΑU
     DEP. IMMUNOL., INSTITUTE MICROBIOL., UNIV. LODZ, BANACHA 12/16, 90-237
CS
     LODZ.
     ARCH IMMUNOL THER EXP, (1981 (RECD 1983)) 29 (6), 805-812
SO
     CODEN: AITEAT. ISSN: 0004-069X.
FS
     BIOSIS
     BIOSIS 1983:240354
OS
LΑ
     English
ED
     Entered STN: 20011116
     Last Updated on STN: 20011116
     1983:75994 TOXCENTER
ΑN
     Copyright 2002 BIOSIS
CP
     The influence of 6-metoxy-cumaranon-2-acetic acid and its 4 derivatives
AB
     [6-hydroxy-cumaranon-2-acetic acid, 6-hydroxy-cumaranon-2-(N-4-
     carboxyphenyl-)acetamide, 6-hydroxy-cumaranon-2-(N-4-
     carboxymethylphenyl-) acetamide (HKCMF) and 6-hydroxy-cumaranon-2-(N-4-
     carboxy-3-hydroxy-phenyl-) acetamide (HKCHF)] on humoral and cellular
     immunogenesis was determined in mice. The study of immunosuppressive
     properties included: control of the level of peripheral blood lymphocytes,
     estimation of transplantation immunity, GvH [graft vs. host] reaction, PFC
     [plaque-forming cell] production for SRBCC [sheep erythrocyte] and LPS
     [lipolysaccharide], and the determintion of the number of cells possessing
     receptors for antigen, Fc fragment and complement. Blast transformation
     of lymphocytes stimulated with PHA [phytohemagglutinin] and the cytotoxic
     effect of sensitized lymphocytes were estimated. Results indicate
     immunosuppressive activity of acetamides HKCMF and HKCHF. They lowered
     the level of circulating lymphocytes, the number of cells possessing
     receptors for complement and they hindered PFC production for SRBC and
     LPS. Acetamide HKCHF weakened the cytotoxic activity of lymphocytes
     sensitized to alloantigens.
     The influence of 6-metoxy-cumaranon-2-acetic acid and its 4 derivatives
AB
     [6-hydroxy-cumaranon-2-acetic acid, 6-hydroxy-cumaranon-2-(N-4-
     carboxyphenyl-) acetamide, 6-hydroxy-cumaranon-2-(N-4-
     carboxymethylphenyl-) acetamide (HKCMF) and 6-hydroxy-cumaranon-2-(N-4-
     carboxy-3-hydroxy-phenyl-) acetamide (HKCHF)] on humoral and cellular
     immunogenesis was determined in mice. The study of immunosuppressive.
ST
     Miscellaneous Descriptors
        MOUSE SHEEP LYMPHOCYTO TOXICITY PLAQUE FORMING CELL ERYTHROCYTE 6
        HYDROXY CUMARANON-2-ACETIC-ACID 6 HYDROXY CUMARANON 2-N-4
        CARBOXYPHENYL ACETAMIDE 6 HYDROXY CUMARANON-2 N-4
        CARBOXYMETHYLPHENYL ACETAMIDE 6 HYDROXY CUMARANON 2-N-4
        CARBOXY-3-HYDROXYPHENYL ACETAMIDE IMMUNOLOGIC-DRUG IMMUNO SUPPRESSANT
        PHYTO HEM AGGLUTININ FC RECEPTOR COMPLEMENT GRAFT
        VS. HOST REACTION
L8
     ANSWER 14 OF 61 USPATFULL
AN
       2002:92268 USPATFULL
TI
       Human G-protein Chemokine Receptor HDGNR10
       Rosen, Craig A., Laytonsville, MD, UNITED STATES
IN
       Roschke, Viktor, Rockville, MD, UNITED STATES
       Li, Yi, Sunnyvale, CA, UNITED STATES
       Ruben, Steven M., Olney, MD, UNITED STATES
PΙ
       US 2002048786
                          A1
                               20020425
ΑI
       US 2001-779879
                          A1
                               20010209 (9)
                           20000209 (60)
PRAI
       US 2000-181258P
                           20000309 (60)
       US 2000-187999P
       US 2000-234336P
                           20000922 (60)
·DT
       Utility
FS
       APPLICATION
       STERNE, KESSLER, GOLDSTEIN & FOX PLLC, 1100 NEW YORK AVENUE, N.W., SUITE
LREP
       600, WASHINGTON, DC, 20005-3934
CLMN
       Number of Claims: 61
ECL
       Exemplary Claim: 1
DRWN
       4 Drawing Page(s)
LN.CNT 17969
```

The present invention relates to a novel human protein called Human

AΒ

G-protein Chemokine Receptor (CCR5) HDGNR10, and isolated polynucleotides encoding this protein. The invention is also directed to human antibodies that bind Human G-protein Chemokine Receptor (CCR5) HDGNR10 and to polynucleotides encoding those antibodies. Also provided are vectors, host cells, antibodies, and recombinant methods for producing Human G-protein Chemokine Receptor (CCR5) HDGNR10 and human anti-Human G-protein Chemokine Receptor (CCR5) HDGNR10 antibodies. The invention further relates to diagnostic and therapeutic methods useful for diagnosing and treating diseases, disorders, and/or conditions related to this novel human protein and these novel human antibodies.

- DETD . . . that may be used according to the methods of the invention include, but are not limited to, glutamyl derivatives of benzoic acid mustard alkylating agent, phosphate derivatives of etoposide or mitomycin C, cytosine arabinoside, daunorubisin, and phenoxyacetamide derivatives of doxorubicin.
- DETD . . . Three major families of cell surface antigens can be identified on monocytes: adhesion molecules, molecules involved in antigen presentation, and Fc receptor. Modulation of the expression of MHC class II antigens and other costimulatory molecules, such as B7 and ICAM-1, may result. . .
- L8 ANSWER 15 OF 61 USPATFULL
- AN 2002:88256 USPATFULL
- TI Recombinant alphavirus particles
- IN Dubensky, Jr., Thomas W., Rancho Sante Fe, CA, United States Polo, John M., San Diego, CA, United States Ibanez, Carlos E., San Diego, CA, United States Driver, David A., San Diego, CA, United States
- PA Chiron Corporation, Emeryville, CA, United States (U.S. corporation)
- PI US 6376236 B1 20020423 AI US 1999-236140 19990122 (9)
- RLI Division of Ser. No. US 1995-404796, filed on 15 Mar 1995, now patented, Pat. No. US 6015686 Continuation-in-part of Ser. No. US 1995-376184, filed on 18 Jan 1995, now abandoned Continuation-in-part of Ser. No. US 1994-348472, filed on 30 Nov 1994, now abandoned Continuation-in-part of Ser. No. US 1994-198450, filed on 18 Feb 1994, now abandoned Continuation-in-part of Ser. No. US 1993-122791, filed on 15 Sep 1993,
- now abandoned DT Utility
- FS GRANTED
- EXNAM Primary Examiner: Brusca, John S.
- LREP McMasters, David D., Dollard, Anne S., Blackburn, Robert P.
- CLMN Number of Claims: 11 ECL Exemplary Claim: 1
- DRWN 37 Drawing Figure(s); 30 Drawing Page(s)
- LN.CNT 9308
- AB Disclosed are recombinant alphavirus particles comprising a) an alphavirus vector construct which directs the expression of a heterologous nucleic acid molecule; b) a capsid protein; and c) an envelope glycoprotein from a virus different from said alphavirus vector.
- DETD . . . 89:33, 1992); carboxypeptidase G2, which will cleave the glutamic acid from para-N-bis (2-chloroethyl) aminobenzoyl glutamic acid, thereby creating a toxic benzoic acid mustard; and Penicillin-V amidase, which will convert phenoxyacetabide derivatives of doxorubicin and melphalan to toxic compounds (see generally, Vrudhula et. . .
- DETD . . . proteins that recognize Fc portions of antibodies. Monoclonal antibodies which recognize only preselected target cells are then bound to such Fc receptor-bearing alphavirus vector particles, such that the vector particles bind to and infect only those preselected target cells (for example, tumor. . .
- L8 ANSWER 16 OF 61 USPATFULL AN 2002:85173 USPATFULL

```
IL-17 receptor like molecules and uses thereof
ΤI
       Jing, Shuqian, Thousand Oaks, CA, UNITED STATES
ΙN
ΡI
       US 2002045213
                          A1
                               20020418
ΑI
       US 2001-809567
                          A1
                               20010315 (9)
       Continuation-in-part of Ser. No. US 2000-724460, filed on 28 Nov 2000,
RLI
       PENDING
                           20000316 (60)
PRAI
       US 2000-189816P
DT
       Utility
FS
       APPLICATION
       MARSHALL, O'TOOLE, GERSTEIN, MURRAY & BORUN, 6300 SEARS TOWER, 233 SOUTH
LREP
       WACKER DRIVE, CHICAGO, IL, 60606-6402
CLMN
       Number of Claims: 71
ECL
       Exemplary Claim: 1
DRWN
       4 Drawing Page(s)
LN.CNT 4685
       Novel IL-17 receptor like polypeptides and nucleic acid molecules
AB
       encoding the same. The invention also provides vectors, host cells,
       agonists and antagonists (including selective binding agents), and
       methods for producing IL-17 receptor like polypeptides. Also provided
       for are methods for the treatment, diagnosis, amelioration, or
       prevention of diseases with IL-17 receptor like polypeptides.
DETD
                (1989). When constructed together with a therapeutic protein,
       an Fc domain can provide longer half-life or incorporate such functions
       as Fc receptor binding, protein A binding,
       complement fixation and perhaps even placental transfer. Id. Table II
       summarizes the use of certain Fc.
       . . . agents, hydrophilic polymers (such as polyvinylpyrrolidone),
DETD
       low molecular weight polypeptides, salt-forming counterions (such as
       sodium), preservatives (such as benzalkonium chloride, benzoic
       acid, salicylic acid, thimerosal, phenethyl alcohol,
       methylparaben, propylparaben, chlorhexidine, sorbic acid or hydrogen
       peroxide), solvents (such as glycerin, propylene glycol or.
rs
     ANSWER 17 OF 61 USPATFULL
AN
       2002:84902 USPATFULL
ΤI
       Nucleic acids, proteins and antibodies
IN
       Rosen, Craig A., Laytonsville, MD, UNITED STATES
       Ruben, Steven M., Olney, MD, UNITED STATES
PI .
       US 2002044941
                          Α1
                               20020418
       US 2001-925302
                          A1
                               20010810 (9)
ΑI
       Continuation-in-part of Ser. No. WO 2000-US5918, filed on 8 Mar 2000,
RLI
       UNKNOWN
PRAI
       US 1999-124270P
                           19990312 (60)
DΤ
       Utility
FS
       APPLICATION
       HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
LREP
CLMN
      Number of Claims: 23
ECL
       Exemplary Claim: 1
DRWN
      No Drawings
LN.CNT 21121
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB
       The present invention relates to novel lung cancer related
       polynucleotides, the polypeptides encoded by these polynucleotides
       herein collectively referred to as "lung cancer antigens," and
       antibodies that immunospecifically bind these polypeptides, and the use
       of such lung cancer polynucleotides, antigens, and antibodies for
       detecting, treating, preventing and/or prognosing disorders of the lung,
       including, but not limited to, the presence of lung cancer and lung
       cancer metastases. More specifically, isolated lung cancer nucleic acid
      molecules are provided encoding novel lung cancer polypeptides. Novel
       lung cancer polypeptides and antibodies that bind to these polypeptides
       are provided. Also provided are vectors, host cells, and recombinant and
       synthetic methods for producing human lung cancer polynucleotides,
       polypeptides, and/or antibodies. The invention further relates to
       diagnostic and therapeutic methods useful for diagnosing, treating,
```

preventing and/or prognosing disorders related to the lung, including

lung cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The invention further relates to methods and/or compositions for inhibiting or promoting the production and/or function of the polypeptides of the invention.

```
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
                                                          98
                                                                  98
                                                                       HAPBV45
                                                293
SUMM . . . gi|182474
                                     51
                       >pir|JL0118|JL0118 Fc gamma (IgG) receptor IIa
                       precursor - human >sp|P12318|FCGA HUMAN
                       LOW AFFINITY IMMUNOGLOBULIN GAMMA
                        FC RECEPTOR II-A PRECURSOR (FC-GAMMA
                       RII-A) (FCRII-A) (IGG FC RECEPTOR
       II-A)
                       (CD32)
       540125
                       cyclin H [Homo sapiens] >gi|532561 cyclin H
                              80 1099 95 95 HFPCA09
       qi|536920
                [Homo sapiens] >pir|I38731|I38731 cyclin. . .
      . . . that may be used according to the methods of the invention
SUMM
      include, but are not limited to, glutamyl derivatives of benzoic
       acid mustard alkylating agent, phosphate derivatives of
       etoposide or mitomycin C, cytosine arabinoside, daunorubisin, and
      phenoxyacetamide derivatives of doxorubicin.
       . . . Clin. Invest. 79:1440-1446 (1987)); anticollagenase-serum;
SUMM
       alpha2-antiplasmin (Holmes et al., J. Biol. Chem. 262(4):1659-1664
       (1987)); Bisantrene (National Cancer Institute); Lobenzarit disodium
       (N-(2)-carboxyphenyl-4- chloroanthronilic acid disodium or
       "CCA"; Takeuchi et al., Agents Actions 36:312-316, 1992); Thalidomide;
       Angostatic steroid; AGM-1470; carboxynaminolmidazole; and
       metalloproteinase inhibitors. . .
      . . . that may be used according to the methods of the invention include, but are not limited to, glutamyl derivatives of benzoic
SUMM
       acid mustard alkylating agent, phosphate derivatives of
       etoposide or mitomycin C, cytosine arabinoside, daunorubisin, and
      phenoxyacetamide derivatives of doxorubicin. Drug Screening
       . . Three major families of cell surface antigens can be identified
DETD
       on monocytes: adhesion molecules, molecules involved in antigen
      presentation, and Fc receptor. Modulation of the
       expression of MHC class II antigens and other costimulatory molecules,
       such as B7 and ICAM-1, may result. . .
L8
     ANSWER 18 OF 61 USPATFULL
AN
       2002:81254 USPATFULL
       Tissue plasminogen activator-like protease
TΙ
      Moore, Paul A., Germantown, MD, United States
IN
       Ruben, Steven M., Olney, MD, United States
       Ebner, Reinhard, Gaithersburg, MD, United States
PA
      Human Genome Sciences, Inc., Rockville, MD, United States (U.S.
       corporation)
                       B1 20020416
      US 6372473 B1 20020416
US 1999-411977 19991004 (9)
Continuation-in-part of Ser. No. US 1998-84491, filed on 27 May 1998
PΙ
ΑI
RLI
      US 1997-48000P 19970528 (60)
PRAI
DT
      Utility
FS
      GRANTED
EXNAM
      Primary Examiner: Slobodyansky, Elizabeth
LREP
      Human Genome Sciences, Inc.
CLMN
      Number of Claims: 77
ECL
       Exemplary Claim: 1
DRWN
       8 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 11319
       The present invention relates to a novel t-PALP protein which is a
AΒ
      member of the serine protease family. In particular, isolated nucleic
      acid molecules are provided encoding the human t-PALP protein. t-PALP
      polypeptides are also provided as are vectors, host cells and
```

recombinant methods for producing the same. The invention further

relates to screening methods for identifying agonists and antagonists of t-PALP activity. Also provided are diagnostic methods for detecting circulatory system-related disorders and therapeutic methods for treating circulatory system-related disorders.

```
Clin. Invest. 79:1440-1446, 1987); anticollagenase-serum;
DETD
       alpha2-antiplasmin (Holmes et al., J. Biol. Chem. 262(4):1659-1664,
       1987); Bisantrene (National Cancer Institute); Lobenzarit disodium
       (N-(2)-carboxyphenyl-4-chloroanthronilic acid disodium or
       "CCA"; Takeuchi et al., Agents Actions 36:312-316, 1992); Thalidomide;
       Angostatic steroid; AGM-1470; carboxynaminolmidazole; and
       metalloproteinase inhibitors such.
       . . . that may be used according to the methods of the invention
DETD
       include, but are not limited to, glutamyl derivatives of benzoic
       acid mustard alkylating agent, phosphate derivatives of
       etoposide or mitomycin C, cytosine arabinoside, daunorubisin, and
       phenoxyacetamide derivatives of doxorubicin.
DETD
                Three major families of cell surface antigens can be identified
       on monocytes: adhesion molecules, molecules involved in antigen
       presentation, and Fc receptor. Modulation of the
       expression of MHC class II antigens and other costimulatory molecules,
       such as B7 and ICAM-1, may result. .
L8
     ANSWER 19 OF 61 USPATFULL
       2002:78442 USPATFULL
AN
TΙ
       Nucleic acids, proteins, and antibodies
       Rosen, Craig A., Laytonsville, MD, UNITED STATES
IN
       Ruben, Steven M., Olney, MD, UNITED STATES
       Barash, Steven C., Rockville, MD, UNITED STATES
PΙ
       US 2002042096
                          Α1
                               20020411
       US 2001-764887
                               20010117 (9)
AΙ
                          Α1
                           20000131 (60)
PRAI
       US 2000-179065P
       US 2000-180628P
                           20000204 (60)
       US 2000-214886P
                           20000628 (60)
       US 2000-217487P
                           20000711 (60)
       US 2000-225758P
                           20000814 (60)
                           20000726 (60)
       US 2000-220963P
                           20000711 (60)
       US 2000-217496P
       US 2000-225447P
                           20000814 (60)
       US 2000-218290P
                           20000714 (60)
       US 2000-225757P
                           20000814 (60)
       US..2000-226868P
                           20000822 (60)
       US 2000-216647P
                           20000707 (60)
       US 2000-225267P
                           20000814 (60)
                           20000707 (60)
       US 2000-216880P
       US 2000-225270P
                           20000814 (60)
                           20001208 (60)
       US 2000-251869P
                           20000927. (60)
       US 2000-235834P
       US 2000-234274P
                           20000921 (60)
       US 2000-234223P
                           20000921 (60)
       US 2000-228924P
                           20000830 (60)
       US 2000-224518P
                           20000814 (60)
       US 2000-236369P
                           20000929 (60)
       US 2000-224519P
                           20000814 (60)
                           20000726 (60)
       US 2000-220964P
       US 2000-241809P
                           20001020 (60)
       US 2000-249299P
                           20001117 (60)
       US 2000-236327P
                           20000929 (60)
                           20001020 (60)
       US 2000-241785P
       US 2000-244617P
                           20001101 (60)
       US 2000-225268P
                           20000814 (60)
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20000929 (60)

20001208 (60)

20001208 (60)

20000901 (60)

20000925 (60)

20000901 (60)

US 2000-236368P

US 2000-251856P

US 2000-251868P

US 2000-229344P

US 2000-234997P

US 2000-229343P

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US 2000-229345P
                           20000901 (60)
                           20000901 (60)
       US 2000-229287P
                           20000905 (60)
       US 2000-229513P
                           20000908 (60)
      US 2000-231413P
      US 2000-229509P
                           20000905 (60)
                           20000929 (60)
      US 2000-236367P
      US 2000-237039P
                           20001002 (60)
                           20001002 (60)
      US 2000-237038P
                           20000929 (60)
      US 2000-236370P
                           20001002 (60)
      US 2000-236802P
                           20001002 (60)
       US 2000-237037P
       US 2000-237040P
                           20001002 (60)
                           20001020 (60)
       US 2000-240960P
       US 2000-239935P
                           20001013 (60)
      Utility
      APPLICATION
      HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
      Number of Claims: 24
ECL Exemplary Claim: 1
      No Drawings
LN.CNT 19583
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       The present invention relates to novel liver related polynucleotides and
       the polypeptides encoded by these polynucleotides herein collectively
       known as "liver antigens," and the use of such liver antigens for
       detecting disorders of the liver, particularly the presence of cancer of
       liver and cancer metastases. More specifically, isolated liver
       associated nucleic acid molecules are provided encoding novel liver
      associated polypeptides. Novel liver polypeptides and antibodies that
      bind to these polypeptides are provided. Also provided are vectors, host
       cells, and recombinant and synthetic methods for producing human liver
      associated polynucleotides and/or polypeptides. The invention further
       relates to diagnostic and therapeutic methods useful for diagnosing,
      treating, preventing and/or prognosing disorders related to the liver,
       including cancer of liver tissues, and therapeutic methods for treating
       such disorders. The invention further relates to screening methods for
       identifying agonists and antagonists of polynucleotides and polypeptides
       of the invention. The present invention further relates to methods
       and/or compositions for inhibiting the production and function of the
       polypeptides of the present invention.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
         . . that may be used according to the methods of the invention
       include, but are not limited to, glutamyl derivatives of benzoic
       acid mustard alkylating agent, phosphate derivatives of
       etoposide or mitomycin C, cytosine arabinoside, daunorubisin, and
      phenoxyacetamide derivatives of doxorubicin.
         . . Clin. Invest. 79:1440-1446 (1987)); anticollagenase-serum;
       alpha2-antiplasmin (Holmes et al., J. Biol. Chem. 262(4):1659-1664
       (1987)); Bisantrene (National Cancer Institute); Lobenzarit disodium
       (N-(2)-carboxyphenyl-4-chloroanthronilic acid disodium or
       "CCA"; Takeuchi et al., Agents Actions 36:312-316, 1992); Thalidomide;
      Angostatic steroid; AGM-1470; carboxynaminolmidazole; and
      metalloproteinase inhibitors such.
       . . . that may be used according to the methods of the invention \boldsymbol{\theta}
       include, but are not limited to, glutamyl derivatives of benzoic
       acid mustard alkylating agent, phosphate derivatives of
       etoposide or mitomycin C, cytosine arabinoside, daunorubisin, and
      phenoxyacetamide derivatives of doxorubicin.
         . . Clin. Invest. 79:1440-1446, (1987)); anticollagenase-serum;
       alpha2-antiplasmin (Holmes et al., J. Biol. Chem. 262(4):1659-1664,
       (1987)); Bisantrene (National Cancer Institute); Lobenzarit disodium
       (N-(2)-carboxyphenyl-4-chloroanthronilic acid disodium or
```

DΤ

FS

LREP CLMN

DRWN

AΒ

SUMM

SUMM

SUMM

DETD

. . . Three major families of cell surface antigens can be identified DETD on monocytes: adhesion molecules, molecules involved in antigen

"CCA"; (Takeuchi et al., Agents Actions 36:312-316, (1992)); and

metalloproteinase inhibitors such as BB94.

presentation, and **Fc receptor**. Modulation of the expression of MHC class II antigens and other costimulatory molecules, such as B7 and ICAM-1, may result. . .

```
ANSWER 20 OF 61 USPATFULL
L8
      2002:72627 USPATFULL
AN
      Nucleic, acids, proteins, and antibodies
ΤI
      Rosen, Craig A., Laytonsville, MD, UNITED STATES
ΙN
       Ruben, Steven M., Olney, MD, UNITED STATES
                              20020404
PΙ
      US 2002039764
                         A1
      US 2001-925298
                         Α1
                              20010810 (9)
ΑI
      Continuation-in-part of Ser. No. WO 2000-US5881, filed on 8 Mar 2000,
RLI
      UNKNOWN
                          19990312 (60)
      US 1999-124270P
PRAI
DT
      Utility
FS
      APPLICATION.
      HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
LREP
CLMN
      Number of Claims: 23
      Exemplary Claim: 1
ECL-
DRWN
      No Drawings
LN.CNT 20087
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
      The present invention relates to novel ovarian cancer and/or breast
AΒ
      cancer related polynucleotides, the polypeptides encoded by these
      polynucleotides herein collectively referred to as "ovarian and/or
      breast antigens," and antibodies that immunospecifically bind these
      polypeptides, and the use of such ovarian and/or breast polynucleotides,
      antigens, and antibodies for detecting, treating, preventing and/or
      prognosing disorders of the reproductive system, particularly disorders
      of the ovaries and/or breast, including, but not limited to, the
      presence of ovarian and/or breast cancer and ovarian and/or breast
      cancer metastases. More specifically, isolated ovarian and/or breast
      nucleic acid molecules are provided encoding novel ovarian and/or breast
      polypeptides. Novel ovarian and/or breast polypeptides and antibodies
      that bind to these polypeptides are provided. Also provided are vectors,
      host cells, and recombinant and synthetic methods for producing human
       ovarian and/or breast polynucleotides, polypeptides, and/or antibodies.
      The invention further relates to diagnostic and therapeutic methods
       useful for diagnosing, treating, preventing and/or prognosing disorders
       related to the ovaries and/or breast, including ovarian and/or breast
       cancer, and therapeutic methods for treating such disorders. The
       invention further relates to screening methods for identifying agonists
       and antagonists of polynucleotides and polypeptides of the invention.
       The invention further relates to methods and/or compositions for
       inhibiting or promoting the production and/or function of the
       polypeptides of the invention.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
SUMM . . . that may be used according to the methods of the invention
       include, but are not limited to, glutamyl derivatives of benzoic
       acid mustard alkylating agent, phosphate derivatives of
       etoposide or mitomycin C, cytosine arabinoside, daunorubisin, and
       phenoxyacetamide derivatives of doxorubicin.
         . . Clin. Invest. 79:1440-1446 (1987)); anticollagenase-serum;
SUMM
       alpha2-antiplasmin (Holmes et al., J. Biol. Chem. 262(4):1659-1664
       (1987)); Bisantrene (National Cancer Institute); Lobenzarit disodium
       (N-(2)-carboxyphenyl-4-chloroanthronilic acid disodium or
       "CCA"; Takeuchi et al., Agents Actions 36:312-316, 1992); Thalidomide;
       Angostatic steroid; AGM-1470; carboxynaminolmidazole; and
      metalloproteinase inhibitors such.
SUMM
       . . . that may be used according to the methods of the invention
       include, but are not limited to, glutamyl derivatives of benzoic
       acid mustard alkylating agent, phosphate derivatives of
```

etoposide or mitomycin C, cytosine arabinoside, daunorubisin, and

on monocytes: adhesion molecules, molecules involved in antigen

. . . Three major families of cell surface antigens can be identified

phenoxyacetamide derivatives of doxorubicin.

DETD

presentation, and **Fc receptor**. Modulation of the expression of MHC class II antigens and other costimulatory molecules, such as B7 and ICAM-1, may result. . .

```
ANSWER 21 OF 61 USPATFULL
L8
AN
       2002:66904 USPATFULL
      Fibroblast growth factor-like molecules and uses thereof
ΤI
       Jing, Shuqian, Thousand Oaks, CA, UNITED STATES
IN
      Bass, Michael Brian, Thousand Oaks, CA, UNITED STATES
      Amgen, Inc. (U.S. corporation)
PA
      US 2002037557
                         A1
                               20020328
ΡI
      US 2001-805805
                               20010313 (9)
ΑI
                         Α1
PRAI
      US 2000-188786P
                          20000313 (60)
DT
      Utility
FS
      APPLICATION
LREP
      MCDONNELL BOEHNEN HULBERT & BERGHOFF, 300 SOUTH WACKER DRIVE, SUITE
      3200, CHICAGO, IL, 60606
CLMN
      Number of Claims: 54
      Exemplary Claim: 1
ECL
DRWN
       5 Drawing Page(s)
LN.CNT 3796
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
      The present invention provides Fibroblast Growth Factor-Like (FGF-L)
      polypeptides and nucleic acid molecules encoding the same. The invention
      also provides selective binding agents, vectors, host cells, and methods
       for producing FGF-L polypeptides. The invention further provides
      pharmaceutical compositions and methods for the diagnosis, treatment,
      amelioration, and/or prevention of diseases, disorders, and conditions
      associated with FGF-L polypeptides.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
      . . . 337:525-31. When constructed together with a therapeutic
DETD
      protein, an Fc domain can provide longer half-life or incorporate such
       functions as Fc receptor binding, protein A binding,
      complement fixation, and perhaps even placental transfer. Id. Table II
      summarizes the use of certain Fc.
               agents, hydrophilic polymers (such as polyvinylpyrrolidone),
DETD
      low molecular weight polypeptides, salt-forming counterions (such as
      sodium), preservatives (such as benzalkonium FGF-Loride, benzoic
       acid, salicylic acid, thimerosal, phenethyl alcohol,
      methylparaben, propylparaben, FGF-Lorhexidine, sorbic acid, or hydrogen
      peroxide), solvents (such as glycerin, propylene glycol, or. .
     ANSWER 22 OF 61 USPATFULL
L8
ΑN
       2002:66871 USPATFULL
      IL-17 like molecules and uses thereof
TΙ
      Medlock, Eugene, Westlake Village, CA, UNITED STATES
IN
      Yeh, Richard, Princeton, NJ, UNITED STATES
      Silbiger, Scott M., Woodland Hills, CA, UNITED STATES
      Elliott, Gary S., Thousand Oaks, CA, UNITED STATES
      Nguyen, Hung Q., Thousand Oaks, CA, UNITED STATES
      Jing, Shuqian, Thousand Oaks, CA, UNITED STATES
PI
      US 2002037524
                         A1
                               20020328
      US 2001-886404
                          Α1
                               20010621 (9)
AΙ
      Continuation-in-part of Ser. No. US 2001-810384, filed on 16 Mar 2001,
RLI
      PENDING
PRAI
      US 2001-266159P
                           20010202 (60)
      US 2000-213125P
                           20000622 (60)
DT
      Utility
      APPLICATION
FS
      MARSHALL, O'TOOLE, GERSTEIN, MURRAY & BORUN, 6300 SEARS TOWER, 233 SOUTH
LREP
      WACKER DRIVE, CHICAGO, IL, 60606-6402
CLMN
      Number of Claims: 74
      Exemplary Claim: 1
ECL
DRWN
      26 Drawing Page(s)
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LN.CNT 5737

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

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AB
       Novel IL-17 like polypeptides and nucleic acid molecules encoding the
       same. The invention also provides vectors, host cells, selective binding
       agents, and methods for producing IL-17 like polypeptides. Also provided
       for are methods for the treatment, diagnosis, amelioration, or
       prevention of diseases with IL-17 like polypeptides, agonists, or
       antagonists thereof.
```

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . (1989). When constructed together with a therapeutic protein, an Fc domain can provide longer half-life or incorporate such functions as Fc receptor binding, protein A binding, complement fixation and perhaps even placental transfer. Id. Table II summarizes the use of certain Fc.

DETD . . . agents; hydrophilic polymers (such as polyvinylpyrrolidone); low molecular weight polypeptides; salt-forming counterions (such as sodium); preservatives (such as benzalkonium chloride, benzoic acid, salicylic acid, thimerosal, phenethyl alcohol, methylparaben, propylparaben, chlorhexidine, sorbic acid or hydrogen peroxide); solvents (such as glycerin, propylene glycol or.

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L8
    ANSWER 23 OF 61 USPATFULL
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ΑN 2002:57821 USPATFULL

Therapeutic inhibitor of vascular smooth muscle cells

ΤĮ IN Kunz, Lawrence L., Redmond, WA, United States Klein, Richard A., Edmonds, WA, United States Reno, John M., Brier, WA, United States

PA NeoRx Corporation, Seattle, WA, United States (U.S. corporation)

PΙ US 6358989 20020319 В1

ΑI US 1999-361194 19990726 (9)

Division of Ser. No. US 1997-829685, filed on 31 Mar 1997 RLT. Continuation-in-part of Ser. No. US 1995-450793, filed on 25 May 1995, now patented, Pat. No. US 5811447 Continuation of Ser. No. WO 1996-US2125, filed on 15 Feb 1996 Continuation-in-part of Ser. No. US 1995-389712, filed on 15 Feb 1995 Continuation of Ser. No. US 1993-62451, filed on 13 May 1993, now abandoned

DT Utility FS GRANTED

Primary Examiner: Barts, Samuel EXNAM

LREP Schwegman, Lundenberg, Woessner & Kluth, PA

CLMN Number of Claims: 20 ECL-· Exemplary Claim: 1

30 Drawing Figure(s); 22 Drawing Page(s) DRWN

LN.CNT 5403

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

Methods are provided for inhibiting stenosis or restenosis following vascular trauma in a mammalian host, comprising administering to the host a therapeutically effective dosage of a cytostatic agent and/or cytoskeletal inhibitor so as to biologically stent the traumatized vessel. Also provided is a method to inhibit or reduce vascular remodeling following vascular trauma, comprising administering an effective amount of a cytoskeletal inhibitor. Further provided are pharmaceutical compositions and kits comprising the therapeutic agents. of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

. include inactive ingredients such as cellulose, pregelatinized DETD starch, silicon dioxide, hydroxypropyl methylcellulose, magnesium stearate, microcrystalline cellulose, starch, talc, titanium dioxide, benzoic acid, citric acid, corn starch, mineral oil, polypropylene glycol, sodium phosphate, and zinc stearate, and the like. Hard or soft gelatin.

DETD . . . does not bind to sites in the patient through antigen-specific binding, but instead binds in a non-specific manner, e.g., through Fc receptor binding reticuloendothelial cells, asialo-receptor binding, and by binding to ubiquitin-expressing cells. The irrelevant "blocker" decreases non-specific binding of the therapeutic.

```
ANSWER 24 OF 61 USPATFULL
ΑN
       2002:51012 USPATFULL
       Fc receptor modulators and uses thereof
ΤI
IN
       Baell, Johathan B., Ivanhoe, AUSTRALIA
       Garrett, Thomas P. J., Brunswick, AUSTRALIA
       Hogarth, P. Mark, Williamstown, AUSTRALIA
       Matthews, Barry R., Olinda, AUSTRALIA
       McCarthy, Thomas D., East Malvern, AUSTRALIA
       Pietersz, Geoffrey A., Greensborough, AUSTRALIA
       Ilexus Pty Limited, Victoria, AUSTRALIA (non-U.S. corporation)
PA
PΙ
       US 6355683
                          В1
                                20020312
       us 1999-393598
ΑI
                                19990910 (9)
       US 1998-99855P
PRAI
                           19980911 (60)
       US 1999-131938P
                           19990430 (60)
       US 1999-148479P
                           19990811 (60)
DT
       Utility
FS
       GRANTED
      Primary Examiner: Spivack, Phyllis G.
EXNAM
LREP
       Sheridan Ross P.C.
CLMN
       Number of Claims: 50
       Exemplary Claim: 1
ECL
DRWN
       20 Drawing Figure(s); 20 Drawing Page(s)
LN.CNT 1876
AΒ
       This invention relates to a pharmaceutical composition comprising a
       Fc receptor modulating compound and a pharmaceutically
       acceptable carrier. The present invention also relates to a method for
       treating a variety of diseases using a Fc receptor
       modulating compound.
ΤI
       Fc receptor modulators and uses thereof
       This invention relates to a pharmaceutical composition comprising a
AΒ
       Fc receptor modulating compound and a pharmaceutically
       acceptable carrier. The present invention also relates to a method for
       treating a variety of diseases using a Fc receptor
       modulating compound.
DRWD
       FIGS. 5A-5G show some of the Fc receptor modulating
       compounds including those corresponding to Fc receptor
       modulating activities shown in FIGS. 6-9;
DETD
       The Fc receptor modulating compounds of the present
       invention can also include nucleosides or derivatives thereof.
       Preferably, the nucleosides of the present invention.
DETD
       The Fc receptor modulating compounds of the present
       invention can further include folic acid or its derivatives.
DETD
       The Fc receptor modulating compounds of the present
       invention can allso include peptides which can modulate the interaction
       between Fc receptors and immunoglobulins..
DETD

    been found to be effective in modulating the FcR receptor

       activities. Thus, in another embodiment of the present invention, the
       Fc receptor modulating compound of the present
       invention also includes a compound of the formula: ##STR26##
DETD
       In addition to and/or instead of a rational drug design, other
       Fc receptor modulators can be identified by a
       screening process, where a variety of compounds are tested to determine
       their Fc receptor modulating activity. In this
       manner, a variety of Fc receptor modulators have
       been identified. Thus, compounds of the present invention include
       substituted and unsubstituted benzoic acids, in particular, 4-methyl
       benzoic acid and 3-methyl benzoic
       acid; nucleosides and analogs thereof; and folic acid and its
       derivatives.
DETD
       The compounds of the present invention are Fc receptor
       modulators, e.g., they modulate Fc receptor binding
       of immunoglobulins. Preferably, the compounds of the present invention
       modulate Fc receptors selected from the group consisting of Fc.alpha.R,.
DETD
       This experiment illustrates a synthesis of 1,2-Bis(m-
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L8

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carboxyphenyl)ethane: ##STR28##
DETD
                   . was extracted with EtOAc (3.times.50 mL) and the combined
           organic extracts dried (Na.sub.2SO.sub.4), filtered and concentrated in
           vacuo to give 1,2-bis(m-carboxyphenyl)ethane as a white solid.
           MS (APCI) m/z 269 (M+1, 100%) .sup.13C NMR (50 MHz, d.sub.6-DMSO):
           .delta.38.4, 128.8, 130.3, 131.1, 132.5,.
           This experiment illustrates a synthesis of 3-[(m-carboxyphenyl
DETD
                                                ##STR29##
           )methoxy]benzoic acid:
DETD
           Step 2: Using 3-[(m-bromophenyl)methoxy]bromobenzene and the method
           described in Example 1, step 2 gave 3-[(m-carboxyphenyl
           ) methoxy] -benzoic acid as a white solid. MS (APCI)
           m/z 271 (M.sup.+-1, 100%). .sup.13C NMR (50 MHz, d.sub.6-DMSO):
           .delta.68.3, 114.5, 119.3, 121.5, 127.8,.
DETD
                   . aqueous HCl (1 M, 50 mL). The organic extract was dried
           (Na.sub.2SO.sub.4), filtered and concentrated in vacuo to give 1,3-bis
           (m-carboxyphenyl)-1-propanol as a viscous oil. MS (APCI) m/z
           299 (M.sup.+-1, 100%). .sup.1H NMR (200 MHz, CDCl.sub.3);
           .delta.1.95-2.10, m, 2H; 2.68-2.83, m,.
           This experiment illustrates a synthesis of (S,S)-1,2-bis-(3-
DETD
           carboxyphenyl) ethane-1,2-diol: ##STR34##
                         The above diester (500 mg, 1.5 mmol) was hydrolyzed using the
DETD
           procedure described in Example 6, step 2 to give (S,S)-1,2-bis-(3-
           carboxyphenyl) ethane-1,2-diol as a white solid. MS (APCI) m/z
           301 (M.sup.+-1, 100%). .sup.1H NMR (200 MHz, d.sub.6-DMSO): .delta.3.40,
           bs, 1H; 4.76, s,.
           Step 3: The ester in Step 2 was hydrolyzed using the procedure described
DETD
           in Example 6, step 2 to give 1-[m-(carboxymethyl)phenyl]-2-[m-(
           carboxyphenyl)]ethane as a white solid. MS (APCI) m/z 283
           (M.sup.+-1, 100%). .sup.1H NMR (200 MHz, d.sub.6-DMSO): .delta.2.92, m,
           4H; 3.55, s,.
DETD
           This experiment illustrates Fc receptor modulating
           activity of some of the compounds of the present invention.
           This experiment illustrates a synthesis of N-(3'-carboxyphenyl
DETD
           )-2-(carboxybenzene)sulfonamide: ##STR41##
                    . The above diester (1.0 \text{ g, } 2.75 \text{ mmol}) was hydrolyzed using the
DETD
           procedure described in Example 6, step 2 to provide N-(3'-
           carboxyphenyl) -2-(carboxybenzene) sulfonamide as a white solid.
           MS (CI) m/z 320 (M.sup.+-1, 100%). .sup.13C NMR (50 MHz, d.sub.6-DMSO):
           .delta.168.0, 166.3, 137.3, 135.8, 133.4,.
           This experiment illustrates a synthesis of (3R,4R)-4,5-bis(m-
DETD
           carboxyphenyl)imidazolid-2-one: ##STR44##
DETD
                    . The above diester (68 mg, 0.19 mmol) was hydrolyzed using the
           procedure described in Example 6, step 2 to give (3R,4R)-4,5-bis(m-
           carboxyphenyl)imidazolid-2-one as a white solid. MS
           (electrospray) m/z 327 (M.sup.++1, 100%). .sup.13C NMR (50 MHz,
           d.sub.6-DMSO): .delta.64.3, 127.4, 129.1, 129.3, 131.1,.
DETD
           This experiment illustrates Fc receptor modulating
           activity of a tripeptide and a hexapeptide.
           For each aggregation experiment, a mixture of 50 .mu.L of the Fc
DETD
           receptor agonist, heat aggregated gamma globulin ("HAGG", 200
           .mu.g/mL) or collagen (2 .mu.g/mL) was incubated with 50 .mu.L of
           phosphate buffered.

    And the second of the second of
CLM
           What is claimed is:
           24. A method for inhibiting Fc receptor binding of
           immunoglobulin in a patient comprising administering to such patient a
           pharmaceutically effective amount of a compound of the.
           25. The method of claim 24, wherein said Fc receptor
           is selected from the group consisting of Fc.alpha.R, Fc.epsilon.R,
           Fc.gamma.R and mixtures thereof.
           26. The method of claim 25, wherein said Fc receptor
```

is selected from the group consisting of Fc.gamma.RIIa, Fc.gamma.RIIb,

Fc.gamma.RIIc and mixtures thereof.

```
ΤI
       49 human secreted proteins
       Moore, Paul A., Germantown, MD, UNITED STATES
IN
       Ruben, Steven M., Olney, MD, UNITED STATES
       Olsen, Henrik S., Gaithersburg, MD, UNITED STATES
       Shi, Yanggu, Gaithersburg, MD, UNITED STATES
       Rosen, Craig A., Laytonsville, MD, UNITED STATES
       Florence, Kimberly A., Rockville, MD, UNITED STATES
       Soppet, Daniel R., Centreville, VA, UNITED STATES
       LaFleur, David W., Washington, DC, UNITED STATES
       Endress, Gregory A., Potomac, MD, UNITED STATES
       Ebner, Reinhard, Gaithersburg, MD, UNITED STATES
       Komatsoulis, George, Silver Spring, MD, UNITED STATES
       Duan, Roxanne D., Bethesda, MD, UNITED STATES
       US 2002026040
PI ·
                          A1
                               20020228
ΑI
       US 2001-904615
                          A1
                               20010716 (9)
RLI
       Continuation of Ser. No. US 2000-739254, filed on 19 Dec 2000, PENDING
       Continuation of Ser. No. US 2000-511554, filed on 23 Feb 2000, ABANDONED
       Continuation-in-part of Ser. No. WO 1999-US19330, filed on 24 Aug 1999,
      19980825 (60)
       US 1998-97917P
PRAI
                           19980831 (60)
       US 1998-98634P
DT
       Utility
FS
       APPLICATION
       HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
LREP
CLMN
       Number of Claims: 23
       Exemplary Claim: 1
ECL
DRWN
       No Drawings
LN.CNT 19401
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       The present invention relates to novel human secreted proteins and
AΒ
       isolated nucleic acids containing the coding regions of the genes
       encoding such proteins. Also provided are vectors, host cells,
       antibodies, and recombinant methods for producing human secreted
       proteins. The invention further relates to diagnostic and therapeutic
       methods useful for diagnosing and treating diseases, disorders, and/or
       conditions related to these novel human secreted proteins.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
            . Clin. Invest. 79:1440-1446, 1987); anticollagenase-serum;
SUMM
       alpha2-antiplasmin (Holmes et al., J. Biol. Chem. 262(4):1659-1664,
       1987); Bisantrene (National Cancer Institute); Lobenzarit disodium
       (N-(2)-carboxyphenyl-4-chloroanthronilic acid disodium or
       "CCA"; Takeuchi et al., Agents Actions 36:312-316, 1992); Thalidomide;
       Angostatic steroid; AGM-1470; carboxynaminolmidazole; and
       metalloproteinase inhibitors such.
          . . that may be used according to the methods of the invention
SUMM
       include, but are not limited to, glutamyl derivatives of benzoic
       acid mustard alkylating agent, phosphate derivatives of
       etoposide or mitomycin C, cytosine arabinoside, daunorubisin, and
       phenoxyacetamide derivatives of doxorubicin.
DETD
         . . Three major families of cell surface antigens can be identified
       on monocytes: adhesion molecules, molecules involved in antigen
       presentation, and Fc receptor. Modulation of the
       expression of MHC class II antigens and other costimulatory molecules,
       such as B7 and ICAM-1, may result.
     ANSWER 26 OF 61 USPATFULL
L8
AN
       2002:43612 USPATFULL
ΤI
       Therapeutic inhibitor of vascular smooth muscle cells
IN
       Kunz, Lawrence L., Redmond, WA, UNITED STATES
       Reno, John M., Brier, WA, UNITED STATES
PI
       US 2002025979
                               20020228
                          A1
ΑI
       US 2001-896208
                          Α1
                               20010629 (9)
RLI
       Division of Ser. No. US 1997-829991, filed on 31 Mar 1997, PENDING
       Continuation-in-part of Ser. No. US 1995-450793, filed on 25 May 1995,
       GRANTED, Pat. No. US 5811447 Continuation of Ser. No. US 1993-62451,
       filed on 13 May 1993, ABANDONED Continuation of Ser. No. WO 1996-US2125,
```

```
filed on 15 Feb 1996, UNKNOWN Continuation-in-part of Ser. No. US
       1995-389712, filed on 15 Feb 1995, PENDING
DT
       Utility
FS
       APPLICATION
       SCHWEGMAN, LUNDBERG, WOESSNER & KLUTH, P.A., 1600 TCF TOWER, 121 SOUTH
LREP
       8TH STREET, MINNEAPOLIS, MN, 55402
       Number of Claims: 60
CLMN
       Exemplary Claim: 1
ECL
       22 Drawing Page(s)
DRWN
LN.CNT 5068
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       Methods are provided for inhibiting stenosis or restenosis following
       vascular trauma in a mammalian host, comprising administering to the
       host a therapeutically effective dosage of a cytostatic agent and/or
       cytoskeletal inhibitor so as to biologically stent the traumatized
       vessel. Also provided is a method to inhibit or reduce vascular
       remodeling following vascular trauma, comprising administering an
       effective amount of a cytoskeletal inhibitor. Further provided are
       pharmaceutical compositions and kits comprising the therapeutic agents
       of the invention.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       . . . include inactive ingredients such as cellulose, pregelatinized
DETD
       starch, silicon dioxide, hydroxypropyl methylcellulose, magnesium
       stearate, microcrystalline cellulose, starch, talc, titanium dioxide,
       benzoic acid, citric acid, corn starch, mineral oil,
       polypropylene glycol, sodium phosphate, and zinc stearate, and the like.
       Hard or soft gelatin.
       . . . does not bind to sites in the patient through antigen-specific
DETD
       binding, but instead binds in a non-specific manner, e.g., through
       Fc receptor binding reticuloendothelial cells,
       asialo-receptor binding, and by binding to ubiquitin-expressing cells. The irrelevant "blocker" decreases non-specific binding of the
       therapeutic.
L8
     ANSWER 27 OF 61 USPATFULL
       2002:27135 USPATFULL
ΑN
ΤI
       Beta-like glycoprotein hormone polypeptide and heterodimer
       Paszty, Christopher J.R., Ventura, CA, UNITED STATES
IN
       Cao, Jin, Tarzana, CA, UNITED STATES
       Danilenko, Dimitry M., Thousand Oaks, CA, UNITED STATES
       Gong, Jianhua, Thousand Oaks, CA, UNITED STATES
       Hill, David C., Thousand Oaks, CA, UNITED STATES
PΙ
       US 2002015981
                          A1
                                20020207
ΑI
       US 2001-818954
                          A1
                              20010327 (9)
       Continuation-in-part of Ser. No. US 2000-723970, filed on 27 Nov 2000,
RLI
       PENDING
                           20000424 (60)
PRAI
       US 2000-199211P
                           20000328 (60)
       US 2000-192654P
DT
       Utility
FS
       APPLICATION
       AMGEN INCORPORATED, MAIL STOP 27-4-A, ONE AMGEN CENTER DRIVE, THOUSAND
LREP
       OAKS, CA, 91320-1799
CLMN
       Number of Claims: 99
       Exemplary Claim: 1
ECL
DRWN
       7 Drawing Page(s)
LN.CNT 4778
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AΒ
       Novel .beta.10 polypeptides and heterodimers thereof, and nucleic acid
       molecules encoding the same are disclosed. The invention also provides
       vectors, host cells, selective binding agents, and methods for producing
       .beta.10 polypeptides and heterodimeric forms thereof, specifically
       .alpha.2/.beta.10. Also provided for are methods for the treatment,
       diagnosis, amelioration, or prevention of diseases with .beta.10
       polypeptides and .alpha.2/.beta.10 heterodimers or their respective
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binding agents.

This application is a continuation-in-part of U.S. application Ser. No. 09/723,970, filed Nov. 27, 2000, which claims the benefit of U.S. Provisional Application Ser. No. 60/199,211, filed Apr. 24, 2000, and U.S. Provisional Application Ser. No. 60/192,654, filed Mar. 28, 2000, which are hereby incorporated by reference.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . (1989). When constructed together with a therapeutic protein, an Fc domain can provide longer half-life or incorporate such functions as Fc receptor binding, protein A binding, complement fixation and perhaps even placental transfer. Id. Table II summarizes the use of certain Fc. . .

DETD . . . agents, hydrophilic polymers (such as polyvinylpyrrolidone), low molecular weight polypeptides, salt-forming counterions (such as sodium), preservatives (such as benzalkonium chloride, benzoic acid, salicylic acid, thimerosal, phenethyl alcohol, methylparaben, propylparaben, chlorhexidine, sorbic acid or hydrogen peroxide), solvents (such as glycerin, propylene glycol or. . .

L8 ANSWER 28 OF 61 USPATFULL AN 2002:22131 USPATFULL

TI 18 Human secreted proteins

IN Shi, Yanggu, Gaithersburg, MD, UNITED STATES
Young, Paul E., Gaithersburg, MD, UNITED STATES
Ebner, Reinhard, Gaithersburg, MD, UNITED STATES
Soppet, Daniel R., Centreville, VA, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES

PI US 2002012966 A1 20020131 AI US 2001-768826 A1 20010125 (9)

RLI Continuation-in-part of Ser. No. WO 2000-US22350, filed on 15 Aug 2000, UNKNOWN

PRAI US 1999-148759P 19990816 (60)

DT Utility

FS APPLICATION

LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850

CLMN Number of Claims: 23 ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 18157

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel human secreted proteins and isolated nucleic acids containing the coding regions of the genes encoding such proteins. Also provided are vectors, host cells, antibodies, and recombinant methods for producing human secreted proteins. The invention further relates to diagnostic and therapeutic methods useful for diagnosing and treating diseases, disorders, and/or conditions related to these novel human secreted proteins.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . Clin. Invest. 79:1440-1446, 1987); anticollagenase-serum; alpha2-antiplasmin (Holmes et al., J. Biol. Chem. 262(4):1659-1664, 1987); Bisantrene (National Cancer Institute); Lobenzarit disodium (N-(2)-carboxyphenyl-4-chloroanthronilic acid disodium or "CCA"; Takeuchi et al., Agents Actions 36:312-316, 1992); Thalidomide; Angostatic steroid; AGM-1470; carboxynaminolmidazole; and metalloproteinase inhibitors such. . .

SUMM . . . that may be used according to the methods of the invention include, but are not limited to, glutamyl derivatives of benzoic acid mustard alkylating agent, phosphate derivatives of etoposide or mitomycin C, cytosine arabinoside, daunorubisin, and phenoxyacetamide derivatives of doxorubicin.

DETD . . . Clin. Invest. 79:1440-1446, (1987)); anticollagenase-serum; alpha2-antiplasmin (Holmes et al., J. Biol. Chem. 262(4):1659-1664, (1987)); Bisantrene (National Cancer Institute); Lobenzarit disodium (N-(2)-carboxyphenyl-4-chloroanthronilic acid disodium or "CCA"; (Takeuchi et al., Agents Actions 36:312-316, (1992)); and metalloproteinase inhibitors such as BB94.

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Three major families of cell surface antigens can be identified
DETD
       on monocytes: adhesion molecules, molecules involved in antigen
       presentation, and Fc receptor. Modulation of the
       expression of MHC class II antigens and other costimulatory molecules,
       such as B7 and ICAM-1, may result.
L8
     ANSWER 29 OF 61 USPATFULL
       2002:19196 USPATFULL
ΑN
       Eukaryotic layered vector initiation systems for production of
TΙ
       recombinant proteins
       Dubensky, Jr., Thomas W., Rancho Sante Fe, CA, United States
IN
       Polo, John M., San Diego, CA, United States
       Driver, David A., San Diego, CA, United States
PΑ
       Chiron Corporation, Emeryville, CA, United States (U.S. corporation)
ΡI
       US 6342372
                          В1
                               20020129
ΑI
       US 1999-350399
                               19990708 (9)
RLI
       Continuation of Ser. No. US 1997-931783, filed on 16 Sep 1997, now
       abandoned Division of Ser. No. US 1995-404796, filed on 15 Mar 1995, now
       patented, Pat. No. US 6015686 Continuation-in-part of Ser. No. US
       1995-376184, filed on 20 Jan 1995, now abandoned Continuation-in-part of
       Ser. No. US 1994-348472, filed on 30 Nov 1994, now abandoned
       Continuation-in-part of Ser. No. US 1994-198450, filed on 18 Feb 1994,
       now abandoned Continuation-in-part of Ser. No. US 1993-122791, filed on
       15 Sep 1993, now abandoned
DT
       Utility
FS
       GRANTED
EXNAM
       Primary Examiner: Brusca, John S.
       McMasters, David D., Dollard, Anne S., Blackburn, Robert P.
LREP
       Number of Claims: 14
CLMN
ECL
       Exemplary Claim: 1
       37 Drawing Figure(s); 30 Drawing Page(s)
DRWN
LN.CNT 10217
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       The present invention provides compositions and methods for utilizing
AΒ
       recombinant alphavirus vectors. Also disclosed are compositions and
       methods for making and utilizing eukaryotic layered vector initiation
       systems.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
DETD
       . . . 89:33, 1992); carboxypeptidase G2, which will cleave the
       glutamic acid from para-N-bis (2-chloroethyl) aminobenzoyl glutamic
       acid, thereby creating a toxic benzoic acid mustard;
       and Penicillin-V amidase, which will convert phenoxyacetabide
       derivatives of doxorubicin and melphalan to toxic compounds (see
       generally, Vrudhula et.
            . proteins that recognize Fc portions of antibodies. Monoclonal
DETD
       antibodies which recognize only preselected target cells are then bound
       to such Fc receptor-bearing alphavirus vector
       particles, such that the vector particles bind to and infect only those
       preselected target cells (for example, tumor.
L8
     ANSWER 30 OF 61 USPATFULL
       2002:16896 USPATFULL
AN
ΤI
       Fibroblast growth factor receptor-like molecules and uses thereof
IN
       Saris, Christiaan M., Newbury Park, CA, UNITED STATES
       Mu, Sharon X., Thousand Oaks, CA, UNITED STATES
       Xia, Min, Newbury Park, CA, UNITED STATES
       Boone, Thomas Charles, Newbury Park, CA, UNITED STATES
       Covey, Todd, Moorpark, CA, UNITED STATES
       Amgen, Inc. (U.S. corporation)
PA
PΙ
       US 2002009776
                         A1
                               20020124
                         A1
                               20010322 (9)
ΑI
       US 2001-815108
       US 2000-191379P
                         20000322 (60)
PRAI
DT
       Utility
FS
       APPLICATION
LREP
       MCDONNELL BOEHNEN HULBERT & BERGHOFF, 300 SOUTH WACKER DRIVE, SUITE
       3200, CHICAGO, IL, 60606
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CLMN
           Number of Claims: 56
ECL
           Exemplary Claim: 1
DRWN
           30 Drawing Page(s)
LN.CNT 4217
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
           The present invention provides Fibroblast Growth Factor Receptor-Like
AB
           (FGFR-L) polypeptides and nucleic acid molecules encoding the same. The
           invention also provides selective binding agents, vectors, host cells,
           and methods for producing FGFR-L polypeptides. The invention further
           provides pharmaceutical compositions and methods for the diagnosis,
           treatment, amelioration, and/or prevention of diseases, disorders, and
           conditions associated with FGFR-L polypeptides.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
           . . . 337:525-31. When constructed together with a therapeutic
DETD
           protein, an Fc domain can provide longer half-life or incorporate such
           functions as Fc receptor binding, protein A binding,
           complement fixation, and perhaps even placental transfer. Id. Table II
. . . agents, hydrophilic polymers (such as polyvinylpyrrolidone),
DETD
           low molecular weight polypeptides, salt-forming counterions (such as
           sodium), preservatives (such as benzalkonium FGFR-Loride,
           benzoic acid, salicylic acid, thimerosal, phenethyl
           alcohol, methylparaben, propylparaben, FGFR-Lorhexidine, sorbic acid, or
           hydrogen peroxide), solvents (such as glycerin, propylene glycol, or.
        ANSWER 31 OF 61 USPATFULL
^{18}
           2002:16578 USPATFULL
AN
           Composition and method for treating inflammatory diseases
TΙ
           Boone, Thomas C., Newbury Park, CA, UNITED STATES
IN
           Hershenson, Susan, Newbury Park, CA, UNITED STATES
           Bevilacqua, Michael P., Boulder, CO, UNITED STATES
           Collins, David S., Fishers, IN, UNITED STATES
           Amgen Inc. (U.S. corporation)
PA:
PΙ
           US 2002009454
                                          Α1
                                                   20020124
                                          A1
ΑI
           US 2001-784623
                                                   20010215 (9)
           Division of Ser. No. US 1998-131247, filed on 7 Aug 1998, PENDING
RLI
                                            19970210
           WO 1997-US2131
PRAI
           US 1997-55185P
                                            19970808 (60)
DΤ
           Utility
FS
           APPLICATION
LREP
           Timothy J. Gaul, U.S. Patent Operations/TJG, Dept. 4300, M/S 27-4-A,
           AMGEN, INC., One Amgen Center Drive, Thousand Oaks, CA, 91320-1799
CLMN
           Number of Claims: 20
           Exemplary Claim: 1
ECL
DRWN
           14 Drawing Page(s)
LN.CNT 3525
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
           A protein which exhibits a therapeutic effect on inflammation and is
           useful for treating IL-1-mediated inflammatory diseases, particularly
           diseases of the joint.
                                                  and the community of the property of the contract of the contr
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
           . . . Therapeutic protein products have been constructed using the Fc
SUMM
           domain to provide longer half-life or to incorporate functions such as
           Fc receptor binding, protein A binding, complement
           fixation and placental transfer which all reside in the Fc proteins of
           immunoglobulins. Id. For.
                                                      . . .
           [0044] Modifications may be made to introduce four amino acid
DETD
           substitutions to ablate the Fc receptor binding site
           and the complement (Clq) binding site.
           . . . and pluronics); viscosity; clarity; color; sterility; stability
DETD
           (e.g., sucrose and sorbitol); antioxidants (e.g., sodium sulfite and
           sodium hydrogen-sulfite); preservatives (e.g., benzoic
           acid and salicylic acid); odor of the formulation; flavoring and
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diluting agents; rate of dissolution (e.g., solubilizers or solubilizing

agents such.

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ANSWER 32 OF 61 USPATFULL
L8
       2002:9650 USPATFULL
AN
       Method of suppressing an immune response to a transplanted organ or
ΤI
       tissue by administering an OX-2 protein
       Gorczynski, Reginald M., Willowdale, CANADA
IN
       Transplantation Technologies Inc., Toronto, CANADA (non-U.S.
PA
       corporation)
                                20020115
PΙ
       US 6338851
                           B1
ΑI
       US 2000-570367
                                20000505 (9)
       Continuation of Ser. No. WO 1998-CA1038, filed on 6 Nov 1998
RLI
                            19971107 (60)
PRAI
       US 1997-64764P
DT
       Utility
       GRANTED
FS
       Primary Examiner: Gambel, Phillip; Assistant Examiner: Roark, Jessica H.
EXNAM
       Bereskin & Parr, Gravelle, Micheline
LREP
CLMN
       Number of Claims: 14
                                 .
ಪ್ರಾನ್ಯ ಅರ್ಜು ನೀಡು ಪ್ರಕ್ಷಣಾಗಿ ಕಾರ್ಯವಾಗಿ ಬಿಲ್ಲ ಪ್ರಸಾಯವಾಗು ಮಾರ್ಯವಾಗಿ ಪ್ರಕ್ರೀ ಸಂಪರ್ಧಕ್ಕೆ ಮುಂದು ಸಂಪರ್ಧಕ್ಕೆ ಮುಂದು ಇತ್ತಗಳನ್ನು ಗರ
       Exemplary Claim: 1
ECL
DRWN
       20 Drawing Figure(s); 18 Drawing Page(s)
LN.CNT 3129
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       Methods and compositions for inducing immune suppression are disclosed.
AB
       The methods involve administering an effective amount of an OX-2 protein
       or a nucleic acid encoding an OX-2 protein. The methods are useful in
       preventing graft rejection, fetal loss, autoimmune disease, and
       allergies. Methods and compositions for preventing immune suppression
       are also disclosed. The methods involve administering an effective
       amount of an agent that inhibits OX-2...
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       . . . acetic acid, propionic acid, glycolic acid, lactic acid,
DETD
       pyruvic acid, oxalic acid, succinic acid, malic acid, tartaric acid,
       citric acid, benzoic acid, salicylic acid,
       benzenesulphonic acid, and tolunesulphonic acids.
DETD.
       . . . seen with a murine IgG1 isotype control (BALB/c anti-TNP, clone
       107.3: unpublished), making it unlikely that the band observed was
       Fc receptor.
     ANSWER 33 OF 61 USPATFULL
L8
ΑN
       2002:3850 USPATFULL
TI
       Fibroblast growth factor-like molecules and uses thereof
ΙN
       Itoh, Nobuyuki, Otsu, JAPAN
PΙ
       US 2002001825
                           Αĺ
                                20020103
ΑI
       US 2001-822485
                           A1
                                20010402 (9)
RLI
       Continuation-in-part of Ser. No. US 2000-540118, filed on 31 Mar 2000,
       PENDING
DT '
       Utility
FS
       APPLICATION
LREP
       FINNEGAN, HENDERSON, FARABOW,, GARRETT & DUNNER, L.L.P., 1300 I Street,
       N.W., Washington, DC, 20005
       Number of Claims: 45
CLMN
ECL
       Exemplary Claim: 1
DRWN
       6 Drawing Page(s)
LN.CNT 4409
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB
       Novel FGF-like polypeptides and nucleic acid molecules encoding the
       same. The invention also provides vectors, host cells, selective binding
       agents, and methods for producing FGF-like polypeptides. Also provided
       for are methods for the treatment, diagnosis, amelioration, or
       prevention of diseases with FGF-like polypeptides.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       . . . (1989). When constructed together with a therapeutic protein,
DETD
```

an Fc domain can provide longer half-life or incorporate such functions

complement fixation and perhaps even placental transfer. Id. Table II

as Fc receptor binding, protein A binding,

summarizes the use of certain Fc.

DETD . . . agents, hydrophilic polymers (such as polyvinylpyrrolidone), low molecular weight polypeptides, salt-forming counterions (such as sodium), preservatives (such as benzalkonium chloride, benzoic acid, salicylic acid, thimerosal, phenethyl alcohol, methylparaben, propylparaben, chlorhexidine, sorbic acid or hydrogen peroxide), solvents (such as glycerin, propylene glycol or . . .

L8 ANSWER 34 OF 61 USPATFULL

AN 2001:231292 USPATFULL

TI Substituted imidazolidine derivatives, their preparation, their use and pharmaceutical preparations including them

IN Wehner, Volkmar, Sandberg, Germany, Federal Republic of Stilz, Hans Ulrich, Frankfurt, Germany, Federal Republic of Schmidt, Wolfgang, Frankfurt, Germany, Federal Republic of Seiffge, Dirk, Mainz-Kostheim, Germany, Federal Republic of

PA Aventis Pharma Deutschland GmbH, Frankfurt am Main, Germany, Federal Republic of (non-U.S. corporation)

AI US 2000-516587 20000301 (9)

RLI Continuation of Ser. No. US 1998-195440, filed on 18 Nov 1998, now abandoned

PRAI DE 1997-19751251 19971119

DT Utility

FS GRANTED

EXNAM Primary Examiner: Oswecki, Jane C. LREP Heller Ehrman White & McAuliffe LLP

CLMN Number of Claims: 33 ECL Exemplary Claim: 1 DRWN No Drawings

LN.CNT 5731

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Substituted imidazolidine derivatives of the formula I, ##STR1##

in which B, E, W, Y, R, R.sup.2, R.sup.3, R.sup.30, e and h have the meanings indicated in the claims. The compounds of the formula I are valuable pharmaceutical active compounds, which are suitable, for example, for the therapy of inflammatory disorders, for example of rheumatoid arthritis, or of allergic disorders. The compounds of the formula I are inhibitors of the adhesion and migration of leucocytes and/or antagonists of the adhesion receptor VLA-4 belonging to the integrins group. They are generally suitable for the therapy or prophylaxis of illnesses which are caused by an undesired extent of leucocyte adhesion and/or leucocyte migration or are associated therewith, or in which cell-cell or cell-matrix interactions which are based on interactions of VLA-4 receptors with their ligands play a part. The invention furthermore relates to processes for the preparation of the compounds of the formula I, their use, in particular as pharmaceutical active compounds, and pharmaceutical preparations which contain compounds of the formula I.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . acid or phosphoric acid, and with organic carboxylic acids or sulfonic acids, such as, for example, acetic acid, citric acid, benzoic acid, maleic acid, fumaric acid, tartaric acid, methanesulfonic acid or p-toluenesulfonic acid. Compounds which contain both acidic groups and basic groups . .

DETD 2.4 The plates were incubated at room temperature for 20 minutes with 100 .mu.l/well of Fc receptor blocking buffer (1 mg/ml of .gamma.-globulin, 100 mM NaCl, 100 .mu.M MgCl.sub.2, 100 .mu.M MnCl.sub.2, 100 .mu.M CaCl.sub.2, 1 mg/ml of BSA in 50 mM HEPES, pH 7.5). After removal of the Fc receptor blocking buffer, washing was carried out once with PBS.

DETD 2.6 U937 cells were incubated in **Fc receptor** blocking buffer for 20 minutes and then added by pipette in a concentration of 1.times.10.sup.6 /ml and in an amount. . .

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L8
     ANSWER 35 OF 61 USPATFULL
AN
       2001:184866 USPATFULL
       Therapeutic inhibitor of vascular smooth muscle cells
ΤI
       Kunz, Lawrence L., Redmond, WA, United States
TN
       Reno, John M., Brier, WA, United States
       NeoRx Corporation, Seattle, WA, United States (U.S. corporation)
PΑ
                          В1
                               20011023
PΙ
       US 6306421
                               19970331 (8)
ΑI
       US 1997-829991
       Continuation-in-part of Ser. No. US 1995-450793, filed on 25 May 1995,
RLI
       now patented, Pat. No. US 5811447 Continuation of Ser. No. US
       1993-62451, filed on 13 May 1993, now abandoned Continuation-in-part of
       Ser. No. US 1993-11669, filed on 28 Jan 1993 Continuation-in-part of
       Ser. No. WO 1992-US8220, filed on 25 Sep 1992 Continuation-in-part of
       Ser. No. WO 1996-US2125, filed on 15 Feb 1996 Continuation-in-part of
       Ser. No. US 1995-389712, filed on 15 Feb 1995, now abandoned
DT
       Utility
       GRANTED
FS
EXNAM
       Primary Examiner: Barts, Samuel
       Schwegman, Lundberg, Woessner & Kluth, P.A.
LREP
CLMN
       Number of Claims: 36
ECL
       Exemplary Claim: 1
       30 Drawing Figure(s); 22 Drawing Page(s)
DRWN
LN.CNT 5649
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       Methods are provided for inhibiting stenosis or restenosis following
AΒ
       vascular trauma in a mammalian host, comprising administering to the
       host a therapeutically effective dosage of a cytostatic agent and/or
       cytoskeletal inhibitor so as to biologically stent the traumatized
       vessel. Also provided is a method to inhibit or reduce vascular
       remodeling following vascular trauma, comprising administering an
       effective amount of a cytoskeletal inhibitor. Further provided are
       pharmaceutical compositions and kits comprising the therapeutic agents
       of the invention.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
      . . . include inactive ingredients such as cellulose, pregelatinized
DETD
       starch, silicon dioxide, hydroxypropyl methylcellulose, magnesium
       stearate, microcrystalline cellulose, starch, talc, titanium dioxide,
       benzoic acid, citric acid, corn starch, mineral oil,
       polypropylene glycol, sodium phosphate, and zinc stearate, and the like.
       Hard or soft gelatin.
            . does not bind to sites in the patient through antigen-specific
DETD
       binding, but instead binds in a non-specific manner, e.g., through
       Fc receptor binding reticuloendothelial cells,
       asialo-receptor binding, and by binding to ubiquitin-expressing cells.
       The irrelevant "blocker" decreases non-specific binding of the
       therapeutic.
L8
     ANSWER 36 OF 61 USPATFULL
ΑN
       2001:165439 USPATFULL
TΙ
       Method to enhance the immunogenicity of an antigen
       Cowing, Carol O., Del Mar, CA, United States
IN
PΙ
       US 2001024649
                         A1
                             20010927
ΑI
       US 2001-809158
                          Α1
                               20010315 (9)
       Continuation-in-part of Ser. No. US 1998-176044, filed on 20 Oct 1998,
RLI
       GRANTED, Pat. No. US 6210672
DT
       Utility
       APPLICATION
FS
       KNOBBE MARTENS OLSON & BEAR LLP, 620 NEWPORT CENTER DRIVE, SIXTEENTH
LREP
       FLOOR, NEWPORT BEACH, CA, 92660
       Number of Claims: 57
CLMN
ECL
       Exemplary Claim: 1
DRWN
       14 Drawing Page(s)
LN.CNT 1919
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       The present invention is related to a method for enhancing the
AΒ
       immunogenicity of an antigen in a mammal by introducing into the mammal
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an antigen or a portion thereof and administering to the mammal a treatment that increases antigen presentation in a lymphoid organ.

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CAS INDEXING IS AVAILABLE FOR THIS PATENT.
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SUMM . . . the lipophilic molecule may be selected from the group consisting of dibutyl phthalate, dibutyl-D-tartarate, N,N-diethyl-toluamide, dibutylfumarate, di(2-ethylhexyl)fumarate, diisooctylmaleate, diethylhexylmaleate, diisooctylfumarate, benzoic acid, bihenylmaleate, dioctylphthalate, dibutylmaleate, dioctymaleate, dibutylsuccinate, dioctylsuccinate, dinonylphthalate, diisononylphthalate, dimethylphthalate, diethylphthalate, diethylmethylphthalate, dibenzylbutylphthalate, diethylmethylphthalate and camphor.

DETD . . . Abbreviation Compound

DBP dibutyl phthalate DBT dibutyl-D-tartarate DET N, N-diethyl-toluamide DBF" dibutylfumarate di(2-ethylhexyl) fumarate DEHF DIOM diisooctylmaleate di(ethylhexyl)maleate **DEHM** DIOF disooctylfumarate benzoic acid BA Ċ camphor bihenylmaleate BMdioctylphthalate DOP DBM dibutylmaleate DOM dioctylmaleate DBS dibutylsuccinate dioctylsuccinate DOS DNP dinonylphthalate DINP diisononylphthalate DMP dimethylphthalate

DETD . . . mixture of isotype control mAbs (rat IgG2a, rat IgG2b, mouse IgG1 and mouse IgG2a, each at 100-200 .mu.g/ml) and the anti-Fc receptor mAb 2.4G2. Langerhans cells were identified by their characteristic light scatter properties (high forward, moderate side scatter) and exceptionally high. . .

CLM What is claimed is:

- wherein the lipophilic molecule is selected from the group consisting of dibutyl phthalate, dibutyl-D-tartarate, N,N-diethyl-toluamide, dibutylfumarate, di(2-ethylhexyl)fumarate, diisooctylmaleate, diethylhexylmaleate, diisooctylfumarate, benzoic acid, bihenylmaleate, dioctylphthalate, dibutylmaleate, dioctymaleate, dibutylsuccinate, dioctylsuccinate, dinonylphthalate, diisononylphthalate, dimethylphthalate, diethylphthalate, dipropylphthalate, diphenylphthalate, dibenzylbutylphthalate, diethylmethylphthalate and camphor.
- . wherein the lipophilic molecule is selected from the group consisting of dibutyl phthalate, dibutyl-D-tartarate, N,N-diethyl-toluamide, dibutylfumarate, di(2-ethylhexyl)fumarate, diisooctylmaleate, diethylhexylmaleate, diisooctylfumarate, benzoic acid, bihenylmaleate, dioctylphthalate, dibutylmaleate, dioctymaleate, dibutylsuccinate, dioctylsuccinate, dinonylphthalate, diisononylphthalate, dimethylphthalate, diethylphthalate, dipropylphthalate, diphenylphthalate, dibenzylbutylphthalate, diethylmethylphthalate and camphor.

L8 ANSWER 37 OF 61 USPATFULL

AN 2001:162845 USPATFULL

TI Composition and method for treating inflammatory diseases

IN Boone, Thomas C., Newbury Park, CA, United States Hershenson, Susan, Newbury Park, CA, United States

```
Bevilacqua, Michael P., Boulder, CO, United States
                  Collins, David S., Fishers, IN, United States
PA
                  Amgen Inc., Thousand Oaks, CA, United States (U.S. corporation)
 PΙ
                  US 6294170
                                                                            20010925
                                                               В1
                  US 1998-131247
                                                                            19980807 (9)
ΑI
PRAI
                  US 1997-55185P
                                                                  19970808 (60)
                 Utility
DT
FS
                  GRANTED
                 Primary Examiner: Born, Michael
EXNAM
                  Gaul, Timothy J., Levy, Ron K., Odre, Steven M.
LREP
                  Number of Claims: 15
CLMN
ECL
                  Exemplary Claim: 1
DRWN
                  14 Drawing Figure(s); 14 Drawing Page(s)
LN.CNT 3022
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
                 A protein which exhibits a therapeutic effect on inflammation and is
                  useful for treating IL-1-mediated inflammatory diseases, particularly
                  diseases of the joint.
                                                                                                the control of the co
                     The same and the same of the s
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
SUMM
                 Therapeutic protein products have been constructed using the Fc domain
                  to provide longer half-life or to incorporate functions such as
                 Fc receptor binding, protein A binding, complement
                  fixation and placental transfer which all reside in the Fc proteins of
                 immunoglobulins. Id. For.
                 Modifications may be made to introduce four amino acid substitutions to
DETD
                 ablate the Fc receptor binding site and the
                 complement (Clq) binding site.
DETD
                  . . and pluronics); viscosity; clarity; color; sterility; stability
                  (e.g., sucrose and sorbitol); antioxidants (e.g., sodium sulfite and
                 sodium hydrogen-sulfite); preservatives (e.g., benzoic
                 acid and salicylic acid); odor of the formulation; flavoring and
                 diluting agents; rate of dissolution (e.g., solubilizers or solubilizing
                 agents such.
L8
            ANSWER 38 OF 61 USPATFULL
                 2001:155766 USPATFULL
ΑN
TΙ
                 49 human secreted proteins
IN
                 Moore, Paul A., Germantown, MD, United States
                 Ruben, Steven M., Oley, MD, United States
                 Olsen, Henrik S., Gaithersburg, MD, United States
                 Shi, Yanggu, Gaithersburg, MD, United States
                 Rosen, Craig A., Laytonsville, MD, United States
                 Florence, Kimberly A., Rockville, MD, United States
                 Soppet, Daniel R., Centreville, VA, United States
                 Lafleur, David W., Washington, DC, United States
                 Endress, Gregory A., Potomac, MD, United States
                 Ebner, Reinhard, Gaithersburg, MD, United States
                 Komatsoulis, George, Silver Spring, MD, United States
                 Duan, Roxanne D., Bethesda, MD, United States
                 US 2001021700
PΤ
                                                              A1
                                                                           20010913
AΙ
                 US 2000-739254
                                                              Α1
                                                                           20001219 (9)
                Continuation of Ser. No. US 2000-511554, filed on 23 Feb 2000, ABANDONED
RLI
                 Continuation-in-part of Ser. No. WO 1999-US19330, filed on 24 Aug 1999,
                 UNKNOWN
PRAI
                 US 1998-97917P
                                                                 19980825 (60)
                 US 1998-98634P
                                                                 19980831 (60)
DT
                 Utility
FS
                 APPLICATION
LREP
                HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN
                Number of Claims: 23
ECL
                 Exemplary Claim: 1
DRWN
                No Drawings
LN.CNT 15462
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
                 The present invention relates to novel human secreted proteins and
                 isolated nucleic acids containing the coding regions of the genes
```

encoding such proteins. Also provided are vectors, host cells, antibodies, and recombinant methods for producing human secreted proteins. The invention further relates to diagnostic and therapeutic methods useful for diagnosing and treating diseases, disorders, and/or conditions related to these novel human secreted proteins.

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CAS INDEXING IS AVAILABLE FOR THIS PATENT.
```

SUMM . . . Clin. Invest. 79:1440-1446, 1987); anticollagenase-serum; alpha2-antiplasmin (Holmes et al., J. Biol. Chem. 262(4):1659-1664, 1987); Bisantrene (National Cancer Institute); Lobenzarit disodium (N-(2)-carboxyphenyl-4-chloroanthronilic acid disodium or "CCA"; Takeuchi et al., Agents Actions 36:312-316, 1992); Thalidomide; Angostatic steroid; AGM-1470; carboxynaminolmidazole; and metalloproteinase inhibitors such.

SUMM . . . that may be used according to the methods of the invention include, but are not limited to, glutamyl derivatives of benzoic acid mustard alkylating agent, phosphate derivatives of etoposide or mitomycin C, cytosine arabinoside, daunorubisin, and phenoxyacetamide derivatives of doxorubicin.

DETD . . . Three major families of cell surface antigens can be identified on monocytes: adhesion molecules, molecules involved in antigen presentation, and **Fc receptor**. Modulation of the expression of MHC class II antigens and other costimulatory molecules, such as B7 and ICAM-1, may result. . .

L8 ANSWER 39 OF 61 USPATFULL

AN 2000:28133 USPATFULL
TI Heterocyclic compounds, their preparation and their use as leucocyte

adhesion inhibitors and VLA-4-antagonists
Wehner, Volkmar, Sandberg, Germany, Federal Republic of

Stilz, Hans Ulrich, Frankfurt, Germany, Federal Republic of Schmidt, Wolfgang, Frankfurt, Germany, Federal Republic of Seiffge, Dirk, Mainz-Kostheim, Germany, Federal Republic of

PA Hoechst Marion Roussel Deutschland GmbH, Frankfurt am Main, Germany, Federal Republic of (non-U.S. corporation)

PI US 6034238 20000307

AI US 1998-158772 19980923 (9)

PRAI DE 1997-19741873 19970923

DT Utility FS Granted

IN

EXNAM Primary Examiner: Higel, Floyd D.

LREP Foley & Lardner
CLMN Number of Claims: 15
ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 2807

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

Compounds of the formula I, ##STR1## in which B, E, W, Y, Z, R, R.sup.2, R.sup.2a, R.sup.2b, R.sup.3, g and h have the meanings indicated in the specifications. The compounds of the formula I are valuable pharmaceutical active compounds, which are suitable, for example, for the therapy and prophylaxis of inflammatory disorders, for example of rheumatoid arthritis, or of allergic disorders. The compounds of the formula I are inhibitors of the adhesion and migration of leucocytes and/or antagonists of the adhesion receptor VLA-4 belonging to the integrins group. They are generally suitable for the therapy or prophylaxis of illnesses which are caused by an undesired extent of leucocyte adhesion and/or leucocyte migration or are associated therewith, or in which cell-cell or cell-matrix interactions which are based on interactions of VLA-4 receptors with their ligands play a part. The invention furthermore relates to processes for the preparation of the compounds of the formula I, their use in the therapy and prophylaxis of the disease states mentioned and pharmaceutical preparations which contain the compounds of the formula I.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . sulfuric acid, nitric acid, methanesulfonic acid,

```
p-toluenesulfonic acid, naphthalenedisulfonic acids, oxalic acid, acetic
       acid, tartaric acid, lactic acid, salicylic acid, benzoic
       acid, formic acid, propionic acid, pivalic acid, diethylacetic
       acid, malonic acid, succinic acid, pimelic acid, fumaric acid, maleic
       acid, malic acid,.
       2.4 The plates were incubated at room temperature for 20 minutes with
DETD
       100 .mu.l/well of Fc receptor blocking buffer (1
       mg/ml of .gamma.-globulin, 100 mM NaCl, 100 .mu.M MgCl.sub.2, 100 .mu.M
       MnCl.sub.2, 100 .mu.M CaCl.sub.2, 1 mg/ml of BSA in 50 mM HEPES, pH
       7.5). After removal of the Fc receptor blocking
       buffer washing was carried out once with PBS.
       2.6 U937 cells were incubated in Fc receptor
DETD
       blocking buffer for 20 minutes and then added by pipette in a
       concentration of 1.times.10.sup.6 /ml and in an amount.
     ANSWER 40 OF 61 USPATFULL
rac{1}{8}
       2000:7195 USPATFULL
ΑN
ΤI
       Method for stimulating an immune response utilizing recombinant
       alphavirus particles
       Dubensky, Jr., Thomas W., Rancho Sante Fe, CA, United States
IN
       Polo, John M., San Diego, CA, United States
       Chang, Steven M.W., San Diego, CA, United States
       Jolly, Douglas J., Leucadia, CA, United States
       Chiron Corporation, Emeryville, CA, United States (U.S. corporation)
PΑ
                               20000118
       US 6015694
ΡI
       US 1997-931869
                               19970916 (8)
AI:
       Division of Ser. No. US 1995-404796, filed on 15 Mar 1995 which is a
RLI
       continuation-in-part of Ser. No. US 1995-376184, filed on 18 Jan 1995,
       now abandoned which is a continuation-in-part of Ser. No. US
       1994-348472, filed on 30 Nov 1994, now abandoned which is a
       continuation-in-part of Ser. No. US 1994-198450, filed on 18 Feb 1994,
       now abandoned which is a continuation-in-part of Ser. No. US
       1993-122791, filed on 15 Sep 1993, now abandoned
DT
       Utility
FS
       Granted
      Primary Examiner: Brusca, John S.
EXNAM
       McMasters, David D., Blackburn, Robert P.
LREP
CLMN
       Number of Claims: 11
ECL
       Exemplary Claim: 1
       35 Drawing Figure(s); 30 Drawing Page(s)
DRWN
LN.CNT 10431
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       The present invention provides compositions and methods for utilizing
AB
       recombinant alphavirus vectors. Also disclosed are compositions and
       methods for making and utilizing eukaryotic layered vector initiation
       systems.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
DETD
       . . . 89:33, 1992); carboxypeptidase G2, which will cleave the
       glutamic acid from para-N-bis (2-chloroethyl) aminobenzoyl glutamic
       acid, thereby creating a toxic benzoic acid mustard;
       and Penicillin-V amidase, which will convert phenoxyacetabide
       derivatives of doxorubicin and melphalan to toxic compounds (see
       generally, Vrudhula et. .
       . . . proteins that recognize Fc portions of antibodies. Monoclonal
DETD
       antibodies which recognize only preselected target cells are then bound
       to such Fc receptor-bearing alphavirus vector
       particles, such that the vector particles bind to and infect only those
       preselected target cells (for example, tumor.
     ANSWER 41 OF 61 USPATFULL
L8
ΑN
       2000:7187 USPATFULL
       Eukaryotic layered vector initiation systems
ΤI
       Dubensky, Jr., Thomas W., Rancho Sante Fe, CA, United States
ΙN
       Polo, John M., San Diego, CA, United States
       Jolly, Douglas J., Leucadia, CA, United States
```

Driver, David A., San Diego, CA, United States

```
PΙ
                               20000118
       US 1995-404796
                               19950315 (8)
ΑI
       Continuation-in-part of Ser. No. US 1995-376184, filed on 20 Jan 1995,
RLI
       now abandoned which is a continuation-in-part of Ser. No. US
       1994-348472, filed on 30 Nov 1994, now abandoned which is a
       continuation-in-part of Ser. No. US 1994-198450, filed on 18 Feb 1994,
       now abandoned which is a continuation-in-part of Ser. No. US
       1993-122791, filed on 15 Sep 1993, now abandoned
DT
       Utility
FS
       Granted
EXNAM
       Primary Examiner: Ketter, James; Assistant Examiner: Brusca, John S.
       Seed & Berry, Kruse, Norman J., Blackburn, Robert P.
LREP
       Number of Claims: 20
CLMN
ECL
       Exemplary Claim: 1
DRWN
       37 Drawing Figure(s); 30 Drawing Page(s)
LN.CNT 10466
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides compositions and methods for utilizing
       recombinant alphavirus vectors. Also disclosed are compositions and
       methods for making and utilizing eukaryotic layered vector initiation
       systems.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       . . . 89:33, 1992); carboxypeptidase G2, which will cleave the
DETD
       glutamic acid from para-N-bis (2-chloroethyl) aminobenzoyl glutamic
       acid, thereby creating a toxic benzoic acid mustard;
       and Penicillin-V amidase, which will convert phenoxyacetabide
       derivatives of doxorubicin and melphalan to toxic compounds (see
       generally, Vrudhula et. . .
       . . . proteins that recognize Fc portions of antibodies. Monoclonal
DETD
       antibodies which recognize only preselected target cells are then bound
       to such Fc receptor-bearing alphavirus vector
       particles, such that the vector particles bind to and infect only those
       preselected target cells (for example, tumor.
     ANSWER 42 OF 61 USPATFULL
rs
AN
       1999:160063 USPATFULL
       Heterocycles as inhibitors of leucocyte adhesion and as VLA-4
TΙ
       antagonists
IN
       Stilz, Hans Ulrich, Frankfurt, Germany, Federal Republic of
       Wehner, Volkmar, Sandberg, Germany, Federal Republic of
       Huels, Christoph, Wackernheim, Germany, Federal Republic of
       Seiffge, Dirk, Mainz-Kostheim, Germany, Federal Republic of
       Hoechst Aktiengesellschaft AG, Frankfurt, Germany, Federal Republic of
PA
       (non-U.S. corporation)
PΙ
       US 5998447
                               19991207
       US 1997-972031
ΑI
                               19971117 (8)
       DE 1996-19647382
                           19961115
PRAI
DT
       Utility
FS
       Granted
EXNAM
       Primary Examiner: Richter, Johann; Assistant Examiner: Keating, Dominic...
LREP
       Foley & Lardner
       Number of Claims: 17
CLMN
ECL
      Exemplary Claim: 1
       No Drawings
DRWN
LN.CNT 4819
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       Compounds of the formula I ##STR1## in which B, D, E, R, W, Y, Z, b, c,
AB
       d, e, f, q and have the meanings indicated in the claims, are inhibitors
       of the adhesion and migration of leucocytes and/or antagonists of the
       adhesion receptor VLA-4 which belongs to the group of integrins. The
       invention relates to the use of compounds of the formula I, and of
       pharmaceutical preparations which contain such compounds, for the
       treatment and prophylaxis of diseases which are caused by an un desired
       extent of leucocyte adhesion and/or leucocyte migration or which are
       associated therewith or in which cell--cell or cell-matrix interactions
```

Chiron Viagene, Inc., Emeryville, CA, United States (U.S. corporation)

PΑ

play a part which are based on interactions of VLA-4 receptors with their ligands, for example of inflammatory processes, rheumatoid arthritis or allergic disorders, and it also relates to the use of compounds of the formula I for the production of pharmaceuticals for use in such diseases. It further relates to novel compounds of the formula I.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . sulfuric acid or phosphoric acid, and with organic carboxylic or sulfonic acids, such as, for example, acetic acid, citric acid, benzoic acid, maleic acid, fumaric acid, tartaric acid, methanesulfonic acid or p-toluenesulfonic acid.

DETD 2.4 The plates were incubated at room temperature for 20 minutes with 100 .mu.l/well of Fc receptor blocking buffer (1 mg/ml of .gamma.-globulin, 100 mM NaCl, 100 .mu.M MgCl.sub.2, 100 .mu.M MnCl.sub.2, 100 .mu.M CaCl.sub.2, 1 mg/ml BSA in 50 mM HEPES, pH 7.5). After removing the Fc receptor blocking buffer, washing was carried out once with PBS.

DETD 2.6 U937 cells were incubated in **Fc receptor**blocking buffer for 20 minutes and then pipetted in at a concentration of 1.times.10.sup.6 /ml and in an amount of. . .

L8 ANSWER 43 OF 61 USPATFULL

AN 1999:141975 USPATFULL

Therapeutic inhibitor of vascular smooth muscle cells

IN Kunz, Lawrence L., Redmond, WA, United States Klein, Richard A., Edmonds, WA, United States Reno, John M., Brier, WA, United States

PA NeoRx Corporation, Seattle, WA, United States (U.S. corporation)

PI US 5981568 19991109

AI US 1997-829685 19970331 (8)

RLI Continuation-in-part of Ser. No. US 1995-450793, filed on 25 May 1995, now patented, Pat. No. US 5811447 which is a continuation of Ser. No. US 1993-62451, filed on 13 May 1993, now abandoned And a continuation-in-part of Ser. No. WO 1996-US2125, filed on 15 Feb 1996 which is a continuation-in-part of Ser. No. US 1995-389712, filed on 15 Feb 1995

DT Utility

TΙ

FS Granted

EXNAM Primary Examiner: Barts, Samuel

LREP Schwegman, Lundberg, Woessner & Kluth, P.A.

CLMN Number of Claims: 56 ECL Exemplary Claim: 1

DRWN 30 Drawing Figure(s); 22 Drawing Page(s)

LN.CNT 5553

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods are provided for inhibiting stenosis or restenosis following vascular trauma in a mammalian host, comprising administering to the host a therapeutically effective dosage of a cytostatic agent and/or cytoskeletal inhibitor so as to biologically stent the traumatized vessel. Also provided is a method to inhibit or reduce vascular remodeling following vascular trauma, comprising administering an effective amount of a cytoskeletal inhibitor. Further provided are pharmaceutical compositions and kits comprising the therapeutic agents of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . include inactive ingredients such as cellulose, pregelatinized starch, silicon dioxide, hydroxypropyl methylcellulose, magnesium stearate, microcrystalline cellulose, starch, talc, titanium dioxide, benzoic acid, citric acid, corn starch, mineral oil, polypropylene glycol, sodium phosphate, and zinc stearate, and the like. Hard or soft gelatin. . .

DETD . . . does not bind to sites in the patient through antigen-specific binding, but instead binds in a non-specific manner, e.g., through Fc receptor binding reticuloendothelial cells, asialo-receptor binding, and by binding to ubiquitin-expressing cells.

The irrelevant "blocker" decreases non-specific binding of the therapeutic. . .

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ANSWER 44 OF 61 USPATFULL
L8
AN
       1998:154038 USPATFULL
       Methods of determining chemicals that modulate expression of genes
TI
       associated with cardiovascular disease
       Foulkes, J. Gordon, Huntington Station, NY, United States
IN
       Liechtfried, Franz E., Vienna, Austria
       Pieler, Christian, Vienna, Austria
       Stephenson, John R., Santa Cruz, CA, United States
       Case, Casey C., Lynbrook, NY, United States
       Oncogene Science, Inc., Uniondale, NY, United States (U.S. corporation)
PA
PΙ
       US 5846720
                               19981208
ΑI
       US 1996-700757
                               19960815 (8)
RLI
       Continuation of Ser. No. US 1992-832905, filed on 7 Feb 1992, now
       patented, Pat. No. US 5580722 which is a continuation-in-part of Ser.
       No. US 1990-555196, filed on 18 Jul 1990, now abandoned which is a
       continuation-in-part of Ser. No. US-1989-382712, filed on 18 Jul 1989,
       now abandoned
DT
       Utility
FS
       Granted
      Primary Examiner: Zitomer, Stephanie W.
EXNAM
       White, John P. Cooper & Dunham LLP
LREP
       Number of Claims: 7
CLMN
       Exemplary Claim: 1
ECL
       47 Drawing Figure(s); 42 Drawing Page(s)
DRWN
LN.CNT 3998
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       The invention provided for a method of transcriptionally modulating the
AΒ
       expression of a gene encoding a protein of interest associated with
       treatment of one or more symptoms of a cardiovascular disease. Further
       provided is a method of determining whether a molecule not previously
       known to be a modulator of protein biosynthesis is capable of directly
       and specifically transcriptionally modulating the expression of a gene
       encoding a protein of interest associated with treatment of one or more
       symptoms of a cardiovascular disease. Screening methods, including
       methods of essentially simultaneously screening molecules to determine
       whether the molecules are capable of directly and specifically
       transcriptionally modulating one or more genes encoding proteins of
       interest associated with treatment of one or more symptoms of a
       cardiovascular disease, are also provided.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       . . . interest may be involved in the uptake of modified lipoproteins
DETD
       e.g. LDL-R, scavenger receptor, advanced glycosylated end-product
       receptor or macrophage FC receptor. The protein of
       interest may be involved in lipid metabolism e.g. AMP-activated protein
       kinase, AMP-activated protein kinase kinase, acetyl CoA.
DETD
                       . . pyrazine
1648
           2-[5,6-Bis(4-sulfo-
                          0.86
                                  7.69 1.00
           phenyl)-1,2,4-triazine-
           3-y1]-4-(4-sulfophenyl)-
           pyridine, trisodium salt
           Bis(2,2,2-trifluoroethyl)
1651
                          0.69
                                  3.57 0.70
           (methocarbonyl-methyl)-
           phosphonate
1655
           2,5-Bis(trifluoro-methyl)
                          0.54
                                 4.76 0.81
            benzoic acid
1703
           3-Bromobenzonitrile
                                       0.90
1704
           4-Bromobenzonitrile
                          0.77
                                  4.16 0.94
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1705
            4-Bromobenzophenone
                           0.54
                                   14.28
                                        0.62
                           0.74
                                   8.33 0.94
 1712
            Calcein Blue
 1720
            (15) - (-) - Camphor
                                   4.76.
                           0.65
      ANSWER 45 OF 61 USPATFULL
 rs
 ΑN
        1998:150739 USPATFULL
 ŤΙ
        Alphavirus vector constructs
        Dubensky, Jr., Thomas W., Rancho Sante Fe, CA, United States
 IN
        Polo, John M., San Diego, CA, United States
        Ibanez, Carlos E., San Diego, CA, United States
        Chang, Stephen M. W., San Diego, CA, United States
        Jolly, Douglas J., Leucadia, CA, United States
        Driver, David A., San Diego, CA, United States
        Belli, Barbara A., San Diego, CA, United States
        Chiron Corporation, Emeryville, CA, United States (U.S. corporation)
 PA
- PI-- US: 5843723 - 19981201 - 19981201
 ΑT
        US 1996-739167
                                19961030 (8)
 RLI
        Continuation of Ser. No. US 1995-404796, filed on 20 Mar 1995 which is a
        continuation-in-part of Ser. No. US 1995-376184, filed on 20 Jan 1995,
        now abandoned which is a continuation-in-part of Ser. No. US
        1994-348472, filed on 30 Nov 1994, now abandoned which is a
        continuation-in-part of Ser. No. US 1994-198450, filed on 18 Feb 1994,
        now abandoned which is a continuation-in-part of Ser. No. US
        1993-122791, filed on 15 Sep 1993, now abandoned
 DT
        Utility
 FS
        Granted
        Primary Examiner: Ketter, James; Assistant Examiner: Brusca, John S.
 EXNAM
        McMasters, David D., Kruse, Norman J., Blackburn, Robert P.
 LREP
        Number of Claims: 47
 CLMN
 ECL
        Exemplary Claim: 1
 DRWN
        37 Drawing Figure(s); 30 Drawing Page(s)
 LN.CNT 10318
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.
 AΒ
        The present invention provides compositions and method,, for utilizing
        recombinant alphavirus vectors.
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.
 DETD
        . . . 89:33, 1992); carboxypeptidase G2, which will cleave the
        glutamic acid from para-N-bis (2-chloroethyl) aminobenzoyl glutamic
        acid, thereby creating a toxic benzoic acid mustard;
        and Penicillin-V amidase, which will convert phenoxyacetabide
        derivatives of doxorubicin and melphalan to toxic compounds (see
        generally, Vrudhula et. .
          . . proteins that recognize Fc portions of antibodies. Monoclonal
 DETD
        antibodies which recognize only preselected target cells are then bound
        to such Fc receptor-bearing alphavirus vector
        particles, such that the vector particles bind to and infect only those
        preselected target cells (for example, tumor.
 L8
      ANSWER 46 OF 61 USPATFULL
        1998:119004 USPATFULL
 AN
 TΙ
        Eukaryotic layered vector initiation systems
 IN
        Dubensky, Jr., Thomas W., P.O. Box 675205, Rancho Sante Fe, CA, United
        States 92067
        Polo, John M., 1222 Reed Ave., Number 4, San Diego, CA, United States
        Jolly, Douglas J., 277 Hillcrest Dr., Leucadia, CA, United States 92024
        Driver, David A., 5142 Biltmore St., San Diego, CA, United States 92117
 PΙ
        US 5814482
                                19980929
        US 1996-739158
 ΑI
                                19961030 (8)
        Division of Ser. No. US 1995-404796, filed on 15 Mar 1995 which is a
 RLI
        continuation-in-part of Ser. No. US 1995-376184, filed on 18 Jan 1995,
        now abandoned which is a continuation-in-part of Ser. No. US
        1994-348472, filed on 30 Nov 1994, now abandoned which is a
```

continuation-in-part of Ser. No. US 1994-198450, filed on 18 Feb 1994, now abandoned which is a continuation-in-part of Ser. No. US 1993-122791, filed on 15 Sep 1993, now abandoned DТ Utility FS Granted Primary Examiner: Ketter, James; Assistant Examiner: Brusca, John S. EXNAM Seed & Berry, Kruse, Norman J., Blackburn, Robert P. LREP Number of Claims: 25 Exemplary Claim: 1

CLMN ECL

DRWN 37 Drawing Figure(s); 30 Drawing Page(s)

LN.CNT 10431

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

The present invention provides compositions and methods for utilizing recombinant alphavirus vectors. Also disclosed are compositions and methods for making and utilizing eukaryotic layered vector initiation systems.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD 89:33, 1992); carboxypeptidase G2, which will cleave the glutamic acid from para-N-bis (2-chloroethyl) aminobenzoyl glutamic acid, thereby creating a toxic benzoic acid mustard; and Penicillin-V amidase, which will convert phenoxyacetabide derivatives of doxorubicin and melphalan to toxic compounds (see generally, Vrudhula et.

. . . proteins that recognize Fc portions of antibodies. Monoclonal DETD antibodies which recognize only preselected target cells are then bound to such Fc receptor-bearing alphavirus vector particles, such that the vector particles bind to and infect only those preselected target cells (for example, tumor.

L8 ANSWER 47 OF 61 USPATFULL

AN 1998:91872 USPATFULL

TI Alphavirus structural protein expression cassettes

IN Dubensky, Jr., Thomas W., Rancho Sante Fe, CA, United States Polo, John M., San Diego, CA, United States Ibanez, Carlos E., San Diego, CA, United States Chang, Stephen M. W., San Diego, CA, United States Jolly, Douglas J., Leucadia, CA, United States Driver, David A., San Diego, CA, United States

PA Chiron Corporation, Emeryville, CA, United States (U.S. corporation)

PΙ US 5789245 19980804

ΑI US 1996-741881 19961030 (8)

RLI Division of Ser. No. US 1995-404796, filed on 15 Mar 1995 which is a continuation-in-part of Ser. No. US 1995-376184, filed on 20 Jan 1995, now abandoned which is a continuation-in-part of Ser. No. US 1994-348472, filed on 30 Nov 1994, now abandoned which is a continuation-in-part of Ser. No. US 1994-198450, filed on 18 Feb 1994, now abandoned which is a continuation-in-part of Ser. No. US 1993-122791, filed on 15 Sep 1993, now abandoned

DT Utility FS Granted

Primary Examiner: Ketter, James; Assistant Examiner: Brusca, John S. EXNAM

McMasters, David D., Kruse, Norman J., Blackburn, Robert P. LREP

CLMN Number of Claims: 29 ECL Exemplary Claim: 1

35 Drawing Figure(s); 30 Drawing Page(s) DRWN

LN.CNT 10270

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

The present invention provides compositions and methods for utilizing AB recombinant alphavirus vectors. Also disclosed are compositions and methods for making and utilizing eukaryotic layered vector initiation systems.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . 89:33, 1992); carboxypeptidase G2, which will cleave the glutamic acid from para-N-bis (2-chloroethyl) aminobenzoyl glutamic acid, thereby creating a toxic benzoic acid mustard;

and Penicillin-V amidase, which will convert phenoxyacetabide derivatives of doxorubicin and melphalan to toxic compounds (see generally, Vrudhula et. . .

DETD . . . proteins that recognize Fc portions of antibodies. Monoclonal antibodies which recognize only preselected target cells are then bound to such Fc receptor-bearing alphavirus vector particles, such that the vector particles bind to and infect only those preselected target cells (for example, tumor. . .

L8 ANSWER 48 OF 61 USPATFULL

96:111313 USPATFULL

TI Methods of determining chemicals that modulate transcriptionally

expression of genes associated with cardiovascular disease

IN Foulkes, J. Gordon, Huntington Station, NY, United States
Liechtfried, Franz E., Vienna, Austria
Pieler, Christian, Vienna, Austria
Stephenson, John R., Santa Cruz, CA, United States

Case, Casey C., Lynbrook, NY, United States

PA Oncogene Science, Inc., Uniondale, NY, United States (U.S. corporation)

PI US 5580722 19961203

AI US 1992-832905 19920207 (7)

RLI Continuation-in-part of Ser. No. US 1990-555196, filed on 18 Jul 1990, now abandoned which is a continuation-in-part of Ser. No. US 1989-382712, filed on 18 Jul 1989, now abandoned

DT Utility

AN

FS Granted

EXNAM Primary Examiner: Zitomer, Stephanie W.

LREP White, John P.

CLMN Number of Claims: 7

ECL Exemplary Claim: 1

DRWN 46 Drawing Figure(s); 42 Drawing Page(s)

LN.CNT 4011

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

The invention provided for a method of directly and specifically transcriptionally modulating the expression of a gene encoding a protein of interest associated with treatment of one or more symptoms of a cardiovascular disease such as atherosclerosis, restenosis or hypertension.

Further provided is a method of determining whether a molecule not previously known to be a modulator of protein biosynthesis is capable of directly and specifically transcriptionally modulating the expression of a gene encoding a protein of interest associated with treatment of one or more symptoms of a cardiovascular disease.

Lastly, the invention provides a method of directly and specifically transcriptionally modulating in a human being the expression of a gene encoding a protein of interest associated with treatment of one or more symptoms of a cardiovascular disease, thus ameliorating the disease.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . interest may be involved in the uptake of modified lipoproteins e.g. LDL-R, scavenger receptor, advanced glycosylated end-product receptor or macrophage FC receptor. The protein of interest may be involved in lipid metabolism e.g. AMP-activated protein kinase, AMP-activated protein kinase, acetyl CoA. . .

1651 Bis(2,2,2-trifluoroethyl) 0.69 3.57 0.70

(methocarbonyl-methyl)phosphonate

1655 2,5-Bis(trifluoro-methyl)

```
4.76 0.81
                          0.54
          benzoic acid
1703
        3-Bromobenzonitrile
                         0.76
                                  10.00
                                       0.90
1704
        4-Bromobenzonitrile
                                  4.16 0.94
                         0.77
1705
        4-Bromobenzophenone
                                  14.28
                         0.54
                                       0.62
                                  8.33 0.94
                         0.74
1712
        Calcein Blue
1720
        (15) - (-) - Camphor 0.65
                                  4.76.
L8
     ANSWER 49 OF 61 USPATFULL
AN
       96:58355 USPATFULL
TI
       Benzothiophenes substituted at the 3-carbonyl
ΙN
       Carlson, Donald G., Indianapolis, IN, United States
       Cullinan, George J., Trafalgar, IN, United States
       Fahey, Kennan J., Indianapolis, IN, United States
       Jackson, William T., Indianapolis, IN, United States
       Roehm, Neal W., Zionsville, IN, United States
       Spaethe, Stephen M., Carmel, IN, United States
       Eli Lilly and Company, Indianapolis, IN, United States (U.S.
PA
       corporation)
PΙ
       US 5532382
                               19960702
AT.
       US 1995-402683
                               19950313 (8)
DT
       Utility
FS
       Granted
       Primary Examiner: Raymond, Richard L.; Assistant Examiner: Lambkin,
EXNAM
       Deborah
       Strode, Janelle D., Sales, James J., Boone, David E.
LREP
CLMN
       Number of Claims: 4
       Exemplary Claim: 1
ECL
DRWN
       No Drawings
LN.CNT 712
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       Provided are compounds of the formula II ##STR1## wherein R.sub.6 and
AB
       R.sub.7 are independently hydrogen or C.sub.1 -C.sub.6 alkyl;
       R.sub.5 is naphthyl, substituted nephthyl, or phenyl substituted one to
       three times with C.sub.1 -C.sub.6 alkoxy, C.sub.1 -C.sub.6 alkyl,
       phenyl, or hydroxy; with the proviso that if the phenyl is substituted
       once with hydroxy, it must be further be substituted once or twice with
       C.sub.1 -C.sub.6 alkoxy, C.sub.1 -C.sub.6 alkyl, phenyl or hydroxy, and
       pharmaceutically acceptable salts thereof.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       To 100 mL of methylene chloride was added 2 drops DMF, 3 g (15 mmol) of
DETD
       4-phenyl benzoic acid, and 15 mls of SOC1.sub.2. The
       mixture was heated to reflux for 16 hours then reduced to dryness. To
       the.
DETD
                      Quantity (mg/5 ml)
Ingredient
Active ingredient
                      0.1-1000
Sodium carboxymethyl cellulose
                      50
                                 mq
                      1,25
                                 mq
  Benzoic acid solution 0.10
                                  mT.
Flavor
                      q.v.
Color
                      q.v.
```

mL

Purified water to

DETD . . . No. 45 mesh U.S. sieve and mixed with the sodium carboxymethyl cellulose and syrup to form a smooth paste. The **benzoic**acid solution, flavor, and color are diluted with some of the water and added, with stirring. Sufficient water is then added. . .

```
. measures the production of LTC4 by the cytokine dependent mast
DETD
        cell line MCII, stimulated by cross linking the high affinity Fc
        receptor for IgE (Fc.sub..epsilon. R1).
     ANSWER 50 OF 61 USPATFULL
1.8
        96:38926 USPATFULL
AN
        Benzothiophenes to inhibit leukotrienes
TТ
        Carlson, Donald G., Indianapolis, IN, United States
IN
        Cullinan, George J., Trafalgar, IN, United States
        Fahey, Kennan J., Indianapolis, IN, United States
        Jackson, William T., Indianapolis, IN, United States
        Roehm, Neal W., Zionsville, IN, United States
        Spaethe, Stephen M., Carmel, IN, United States
PΑ
        Eli Lilly and Company, Indianapolis, IN, United States (U.S.
        corporation)
       US 5514704
                                19960507
PT
       US 1995-402598
                                19950313 (8)
AΙ
DT
       Utility
FS Granted
       Primary Examiner: Raymond, Richard L.; Assistant Examiner: Lambkin,
EXNAM
        Strode, Janelle D., Sales, James J., Boone, David E.
LREP
       Number of Claims: 6
CLMN
       Exemplary Claim: 1
ECL
DRWN
       No Drawings
LN.CNT 714
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       A method of inhibiting leukotrienes comprising administering to a mammal
AΒ
        in need thereof an effective amount of a compound having the formula
        ##STR1## wherein R.sub.1 and R.sub.2 are independently hydrogen or
        C.sub.1 -C.sub.6 alkyl;
       R.sub.3 is hydrogen, or a group of the formula ##STR2## wherein R.sub.4
       is phenyl, substituted phenyl, naphthyl or substituted naphthyl, with
       the proviso that when R.sub.1 and R.sub.2 are both C.sub.1 -C.sub.6
        alkyl, R.sub.3 is not hydrogen; and pharmaceutically acceptable salts
        thereof.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       To 100 mL of methylene chloride was added 2 drops DMF, 3 g (15 mmol) of
        4-phenyl benzoic acid, and 15 mls of SOCl.sub.2. The
       mixture was heated to reflux for 16 hours then reduced to dryness. To
        the.
DETD
                      Quantity (mg/5 ml)
Ingredient
                     0.1 - 1000
Active ingredient
Sodium carboxymethyl cellulose
                      50
                      1.25
Syrup
                                 mg
  Benzoic acid solution
                      0.10
                                 mL
Flavor
                      q.v.
Color
                      q.v.
Purified water to
                                 mL
             . No. 45 mesh U.S. sieve and mixed with the sodium carboxymethyl
DETD
        cellulose and syrup to form a smooth paste. The benzoic
       acid solution, flavor, and color are diluted with some of the
       water and added, with stirring. Sufficient water is then added.
DETD
        . . . measures the production of LTC4 by the cytokine dependent mast
        cell line MCII, stimulated by cross linking the high affinity Fc
```

L8 ANSWER 51 OF 61 USPATFULL

AN 96:38925 USPATFULL

TI Benzothiophene compounds useful for inhibiting lipoxygenase

receptor for IgE (Fce.sub..epsilon. R1).

```
Carlson, Donald G., Indianapolis, IN, United States
       Cullinan, George J., Trafalgar, IN, United States
       Fahey, Kennan J., Indianapolis, IN, United States
       Jackson, William T., Indianapolis, IN, United States
       Roehm, Neal W., Zionsville, IN, United States
       Spaethe, Stephen M., Carmel, IN, United States
       Eli Lilly and Company, Indianapolis, IN, United States (U.S.
PA
       corporation)
ΡI
       US 5514703
                               .19960507
ΑI
       US 1995-402593
                               19950313 (8)
DΤ
       Utility
FS
       Granted
       Primary Examiner: Raymond, Richard L.; Assistant Examiner: Lambkin,
EXNAM
       Deborah
       Strode, Janelle D., Sales, James J., Boone, David E.
LREP
       Number of Claims: 5
CLMN
ECL
       Exemplary Claim: 1
DRWN
       No Drawings
LN.CNT 700
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AΒ
       A method of inhibiting 5-lipoxygenase comprising administering to a
       mammal in need thereof an effective amount of a compound having the
       formula ##STR1## wherein R.sub.1 and R.sub.2 are independently hydrogen
       or C.sub.1 -C.sub.6 alkyl;
       R.sub.3 is hydrogen, or a group of the formula ##STR2## wherein R.sub.4
       phenyl, substituted phenyl, naphthyl or substituted naphthyl, with the
       proviso that when R.sub.1 and R.sub.2 are both C.sub.1 -C.sub.6 alkyl,
       R.sub.3 is not hydrogen; and pharmaceutically acceptable salts thereof.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       To 100 mL of methylene chloride was added 2 drops DMF, 3 g (15 mmol) of
DETD
       4-phenyl benzoic acid, and 15 mls of SOC1.sub.2. The
       mixture was heated to reflux for 16 hours then reduced to dryness. To
       the.
DETD
Formulation 8: Suspensions
                     Quantity (mg/5 ml)
Ingredient
Active ingredient
                     0.1-1000
Sodium carboxymethyl cellulose
                     50
                     1.25
                               mg
Syrup
 Benzoic acid solution
                     0.10
                               mL
Flavor
                     q.v.
Color
                     q.v.
Purified water to
                               mL
          . . No. 45 mesh U.S. sieve and mixed with the sodium carboxymethyl
       cellulose and syrup to form a smooth paste. The benzoic
       acid solution, flavor, and color are diluted with some of the
       water and added, with stirring. Sufficient water is then added.
       . . . measures the production of LTC4 by the cytokine dependent mast
DETD
       cell line MCII, stimulated by cross linking the high affinity {f Fc}
       receptor for IgE (Fc.sub..epsilon. R1).
L8
     ANSWER 52 OF 61 USPATFULL
       95:112346 USPATFULL
ΑN
       Cancerous B cell treatment using substituted nucleoside derivatives
ΤI
       Goodman, Michael G., Rancho Santa Fe, CA, United States
IN
       Piro, Lawrence D., La Jolla, CA, United States
       The Scripps Research Institute, La Jolla, CA, United States (U.S.
PA
       corporation)
                               19951219
       US 5476659
PΤ
                               19931112 (8)
       US 1993-151142
ΑI
       Continuation-in-part of Ser. No. US 1992-975830, filed on 13 Nov 1992,
RLI
```

IN

now abandoned which is a continuation-in-part of Ser. No. US 1992-945215, filed on 15 Sep 1992, now patented, Pat. No. US 5317013 which is a division of Ser. No. US 1990-562101, filed on 2 Aug 1990, now patented, Pat. No. US 5147636 which is a division of Ser. No. US 1989-361974, filed on 9 Jun 1989, now patented, Pat. No. US 4948730 which is a division of Ser. No. US 1987-14618, filed on 13 Feb 1987, now patented, Pat. No. US 4849411 which is a continuation of Ser. No. US 1983-546679, filed on 1 Nov 1983, now patented, Pat. No. US 4643992 which is a continuation-in-part of Ser. No. US 1982-439846, filed on 9 Nov 1982, now patented, Pat. No. US 4539205

DT Utility FS Granted

EXNAM Primary Examiner: Kim, Kay K. A.

LREP Welsh & Katz, Ltd. CLMN Number of Claims: 32

ECL Exemplary Claim: 1

DRWN 7 Drawing Figure(s); 7 Drawing Page(s)

LN.CNT 2109

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

Processes for the killing of cancerous B cells, and particularly chronic AB lymphocytic leukemia (CLL) cells are disclosed. In one process, cancerous B cells that do not proliferate when contacted with an immune response-enhancing agent are contacted with an amount of such an agent sufficient to cause peripheral CLL cells to undergo blast transformation and proliferation. The contacted cells are then maintained for a time period sufficient for them to die from that contact. Further contacting of those cells with a cytotoxic amount of an anti-cancer drug or cytotoxic conjugate enhances the death of those cancer cells. In another process, peripheral CLL cells that proliferate on contact with an immune response-enhancing-agent are contacted with a proliferation-inducing amount of such an agent. The contacted cells are maintained for a time period sufficient to undergo blast transformation and proliferation, and the blasts are then contacted with a cytotoxic amount of an anti-cancer drug or cytotoxic conjugate and maintained.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM R.sub.1 contains up to about 20 atoms and has a Hammett substituent sigma constant for ionization of a meta-substituted benzoic acid that is greater than that of hydrogen;

DETD . . . antigens in a dose-dependent manner. Included among those up-regulated antigens are the well-known antigens denominated CD-22, CD-23 (low affinity IgE Fc receptor), CD-25 (IL-2 receptor; p55, Tac), CD-38 and CD-54 (ICAM-1).

DETD With reference to Hammett substituent sigma constants for meta benzoic acid substituents, the preferred 8-substituents have positive values. More preferably, the 8-substituents have sigma constants of about 0.1 to about 0.7.. . .

CLM What is claimed is:

- . ; R.sub.1 contains up to about 20 atoms and has a Hammett substituent sigma constant for ionization of a meta-substituted **benzoic** acid that is greater than that of hydrogen; R.sub.2 is a radical having a length up to about that of an.
- . ; R.sub.1 contains up to about 20 atoms and has a Hammett substituent sigma constant for ionization of a meta-substituted **benzoic** acid that is greater than that of hydrogen; R.sub.2 is a radical having a length up to about that of an. . .
- . ; R.sub.1 contains up to about 20 atoms and has a Hammett substituent sigma constant for ionization of a meta-substituted **benzoic** acid that is greater than that of hydrogen; R.sub.2 is a radical having a length up to about that of an. . .
- L8 ANSWER 53 OF 61 USPATFULL
- AN 94:46970 USPATFULL
- TI Modulation of animal cellular responses with compositions containing 8-substituted guanine derivatives
- IN Goodman, Michael G., Carlsbad, CA, United States Weigle, William O., Del Mar, CA, United States

```
PΙ
       US 5317013
                               19940531
                               19920915 (7)
ΑI
       US 1992-945215
       Division of Ser. No. US 1990-562101, filed on 2 Aug 1990, now patented,
RLI
       Pat. No. US 5147636 which is a division of Ser. No. US 1989-361974,
       filed on 6 Jun 1989, now patented, Pat. No. US 4948730 which is a
       division of Ser. No. US 1987-14618, filed on 13 Feb 1987, now patented,
       Pat. No. US 4849411 which is a continuation of Ser. No. US 1983-546679,
       filed on 1 Nov 1983, now patented, Pat. No. US 4643992 which is a
       continuation-in-part of Ser. No. US 1982-439846, filed on 9 Nov 1982,
       now patented, Pat. No. US 4539205
DΤ
       Utility
FS
       Granted
EXNAM
       Primary Examiner: Hill, Jr., Robert J.; Assistant Examiner: Carlson, K.
       Cochrane
LREP
       Welsh & Katz, Ltd.
CLMN
       Number of Claims: 9
       Exemplary Claim: 1
ECL
       38 Drawing Figure(s); 31 Drawing Page(s)
DRWN
LN.CNT 2169
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       Compositions and methods for their use in modulating animal cellular
       responses are disclosed. The compositions include as an active agent an
       effective amount of an 8-substituted quanine derivative bonded 9-1' to
       an aldose having 5 or 6 carbon atoms in the aldose chain. The
       composition includes a diluent amount of a physiologically tolerable
       carrier. The quanine derivative is free of electrically charged
       functionality, while the 8-substituent has an electron withdrawing
       inductive effect greater than that of hydrogen and contains fewer than
       about 15 atoms. Animal cellular responses are modulated by contacting
       the cells with a composition of this invention.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
DETD
                     . . . Cells
                                           bone marrow-derived lymphocytes
8-BrcGMP
               8-bromoguanosine 3',5'-cyclic
               monophosphate
               8-bromoguanosine .
8-BrGuo
               concanavalin A
Con A
CR
               complement receptor
               guanosine 3',5'-cyclic
cGMP
               monophosphate
FΑ
               erythrocyte-antibody complexes
               erythrocyte-antibody-complement
EAC
               complexes
FcR
               Fc receptor
FCS
               fetal calf serum
Guo
               guanosine
8-HGuo
               8-haloguanosine
[.sup.3 H]TdR tritium labelled
               deoxyribosylthymidine
Ia antigen antigens controlled by the
               immune response genes
IdA, D, E, G and M
               immunoglobulins.
DETD
       With reference to Hammett substituent sigma constants for meta
      benzoic acid substituents, the preferred
       8-substituents have positive values. More preferably, the 8-substituents
       have sigma constants of about 0.1 to about 0.7.. . .
DETD
       . . . interface cells collected separately. Rosettes were lysed in
       0.83% NH.sub.4 Cl, and cells were counted, washed, and used in culture.
       Fc receptor-bearing cells were separated by EA
       rosetting (Parish and Hayward, supra), following the above protocol,
       with the omission of fresh mouse. . .
L8
     ANSWER 54 OF 61 USPATFULL
```

Scripps Clinic and Research Foundation, La Jolla, CA, United States

PA

AN

92:76612 USPATFULL

(U.S. corporation)

```
8-substituted guanine derivatives and interferons
       Goodman, Michael G., Carlsbad, CA, United States
IN
       Weigle, William O., Del Mar, CA, United States
       Scripps Clinic and Research Foundation, La Jolla, CA, United States
PA
       (U.S. corporation)
                               19920915
PΙ
       US 5147636
                               19900802 (7)
ΑI
       US 1990-562101
       Division of Ser. No. US 1989-361974, filed on 6 Jun 1989, now patented,
RLI
       Pat. No. US 4948730 which is a division of Ser. No. US 1987-14618, filed
       on 13 Feb 1987, now patented, Pat. No. US 4849411 which is a
       continuation of Ser. No. US 1983-546679, filed on 1 Nov 1983, now
       patented, Pat. No. US 4643992 which is a continuation-in-part of Ser.
       No. US 1982-439846, filed on 9 Nov 1982, now patented, Pat. No. US
       4539205
DT
       Utility
FS
       Granted
       Primary Examiner: Wax, Robert A.; Assistant Examiner: Ekstrom, Richard
EXNAM
      LREP
       Dressler, Goldsmith, Shore, Sutker & Milnamow, Ltd.
       Number of Claims: 9
CLMN
ECL
       Exemplary Claim: 1
DRWN.
       32 Drawing Figure(s); 31 Drawing Page(s)
LN.CNT 2196
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       Compositions and methods for their use in modulating animal cellular
AB .
       responses are disclosed. The compositions include as an active agent an
       effective amount of an 8-substitued guanine derivative bonded 9-1' to an
       aldose having 5 or 6 carbon atoms in the aldose chain. The composition
       includes a diluent amount of a physiologically tolerable carrier. The
       quanine derivative is free of electrically charged functioinality, while
       the 8-substituent has an electron withdrawing inductive effect greater
       than that of hydrogen and contains fewer than about 15 atoms. Animal
       cellular responses are modulated by contacting the cells with a
       composition of this invention.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
DETD
                          . Cells
                                           bone marrow-derived lymphocytes
               8-bromoguanosine 3',5'-cyclic
8-BrcGMP
               monophosphate
               8-bromoguánosine
8-BrGuo
               concanavalin A
Con A
               complement receptor
CR
               guanosine 3',5'-cyclic
cGMP.
               monophosphate
EΑ
               erythrocyte-antibody complexes
EAC
              erythrocyte-antibody-complement
               complexes
FcR
               Fc receptor
FCS
               fetal calf serum
               guanosine
Guo
8-HGuo
               8-haloguanosine
[.sup.3 H]TdR tritium labelled
               deoxyribosylthymidine
Ia antigen
               antigens controlled by the
               immune response genes
IgA, D, E, G and M
               immunoglobulins.
       With reference to Hammett substituent sigma constants for meta
DETD
       benzoic acid substituents, the preferred
       8-substituents have positive values. More preferably, the 8-substituents
       have sigma constants of about 0.1 to about 0.7.. .
DETD
       . . . interface cells collected separately. Rosettes were lysed in
       0.83% NH.sub.4 Cl, and cells were counted, washed, and used in culture.
       Fc receptor-bearing cells were separated by EA
       rosetting (Parish and Hayward, supra), following the above protocol,
       with the omission of fresh mouse.
```

Modulation of animal cellular responses with compositions containing

TΙ

```
ANSWER 55 OF 61 USPATFULL
L8
ΑN
       91:104199 USPATFULL
       Methods of treating diseases characterized by interactions of
TI
       IgG-containing immune complexes with macrophage Fc receptors using
       antiestrogenic benzothiophenes
       Schreiber, Alan D., Philadelphia, PA, United States
ΙN
       University of Pennsylvania, Philadelphia, PA, United States (U.S.
PA
       corporation)
                               19911224
PΙ
       US 5075321
                               19880224 (7)
       US 1988-159714
ΑI
       Continuation-in-part of Ser. No. US 1987-30028, filed on 24 Mar 1987,
RLI
       now abandoned And a continuation-in-part of Ser. No. US 1987-89790,
       filed on 27 Aug 1987, now patented, Pat. No. US 4902681
DT
       Utility
       Granted
FS
       Primary Examiner: Nutter, Nathan M.
EXNAM
LREP
       Woodcock Washburn Kurtz Mackiewicz & Norris
      Number of Claims: 53
CLMN
ECL
       Exemplary Claim: 1
DRWN
       2 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 641
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       Clearance of antibody-coated cells from the circulation is modulated by
AΒ
       administering an effective amount of certain benzothiophene derivatives,
       or the physiologically acceptable acid addition salts thereof. The
       compounds are useful in treating mammalian diseases characterized by
       interactions between IqG containing immune complexes and macrophage Fc
       receptors.
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       In the spleen, IgG-containing immune complexes bind by the Fc region of
SUMM
       IgG to macrophages at Fc receptor sites on the
       macrophage surface. The Fc portion of the immunoglobulin molecule
       (identified by papain cleavage) is believed to be.
       . . . metaphosphoric acid, succinic acid, formic acid, phthalic acid,
DETD
       lactic acid and the like, most preferably with hydrochloric acid, citric
       acid, benzoic acid, maleic acid, acetic and
       propionic acid. Although the free bases may be used, acid addition
       salts, such as keoxifene HCl,.
     ANSWER 56 OF 61 USPATFULL
^{18}
AN
       90:63453 USPATFULL
ΤI
       Modulation of animal cellular responses with compositions containing
       8-substituted guanine derivatives
       Goodman, Michael G., Carlsbad, CA, United States
ΙN
       Weigle, William O., Del Mar, CA, United States
       Scripps Clinic and Research Foundation, La Jolla, CA, United States
PΑ
       (U.S. corporation)
                               19900814
PΙ
       US 4948730
ΑI
       US 1989-361974
                               19890606 (7)
       Division of Ser. No. US 1987-14618, filed on 13 Feb 1987, now patented,
RLI
       Pat. No. US 4849411 which is a continuation of Ser. No. US 1983-546679,
       filed on 1 Nov 1983, now patented, Pat. No. US 4643992 which is a
       continuation-in-part of Ser. No. US 1982-439846, filed on 9 Nov 1982,
       now patented, Pat. No. US 4539205
DT
       Utility
FS
       Granted
      Primary Examiner: Hazel, Blondel
EXNAM
       Dressler, Goldsmith, Shore, Sutker & Milnamow, Ltd.
LREP
CLMN
       Number of Claims: 7
ECL 
       Exemplary Claim: 1
       38 Drawing Figure(s); 31 Drawing Page(s)
DRWN
LN.CNT 2159
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       Compositions and methods for their use in modulating animal cellular
·AB
```

responses are disclosed. The compositions include as an active agent an

effective amount of an 8-substituted guanine derivative bonded 9-1' to an aldose having 5 or 6 carbon atoms in the aldose chain. The composition includes a diluent amount of a physiologically tolerable carrier. The guanine derivative is free of electrically charged funtionality, while the 8-substituent has an electron withdrawing inductive effector greater than that of hydrogen and contains fewer than about 15 atoms. Anmimal cellular responses are modulated by contacting the cells with a composition of this invention.

```
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
                                          bone marrow-derived lymphocytes
DETD
                     . . . Cells
               8-bromoguanosine 3',5'-cyclic
8-BrcGMP
               monophosphate
               8-bromoguanosine
8-BrGuo
               concanavalin A
Con A
               complement receptor
CR
               guanosine 3',5'-cyclic
cGMP
               monophosphate
             erythrocyte-antibody complexes
EAC
               erythrocyte-antibody-complement
               complexes
               Fc receptor
FcR
FCS
               fetal calf serum
               guanosine
Guo
8-HGuo
               8-haloguanosine
[.sup.3 H]TdR tritium labelled
               deoxyribosylthymidine
Ia antigen
               antigens controlled by the
               immune response genes
IgA, D, E, G and M
               immunoglobulins. .
       With reference to Hammett substituent sigma constants for meta
DETD
       benzoic acid substituents, the preferred
       8-substituents have positive values. More preferably, the 8-substituents
       have sigma constants of about 0.1 to about 0.7.. .
       . . . interface cells collected separately. Rosettes were lysed in
DETD
       0.83% NH.sub.4 Cl, and cells were counted, washed, and used in culture.
       Fc receptor-bearing cells were separated by EA
       rosetting (Parish and Hayward, supra), following the above protocol,
       with the omission of fresh mouse. .
     ANSWER 57 OF 61 USPATFULL
L8
       89:58721 USPATFULL
AN
TI
       Modulation of animal cellular responses with compositions containing
       8-substituted guanine derivatives
       Goodman, Michael G., Carlsbad, CA, United States
IN
       Weigle, William O., Del Mar, CA, United States
       Scripps Clinic and Research Foundation, La Jolla, CA, United States
PΑ
       (U.S. corporation)
PΙ
       US 4849411
                               19890718
ΑI
       US 1987-14618
                               19870213 (7)
DCD
       20020903
       Continuation of Ser. No. US 1983-546679, filed on 1 Nov 1983, now
       patented, Pat. No. US 4643992 which is a continuation-in-part of Ser.
       No. US 1982-439846, filed on 9 Nov 1982, now patented, Pat. No. US
       4539205
DT
       Utility
FŚ.
       Granted
EXNAM Primary Examiner: Hazel, Blondel
       Dressler, Goldsmith, Shore, Sutker & Milnamow, Ltd.
LREP
       Number of Claims: 17
CLMN
       Exemplary Claim: 1
ECL
       32 Drawing Figure(s); 31 Drawing Page(s)
DRWN
LN.CNT 2206
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
       Compositions and methods for their use in modulating animal cellular
AΒ
       responses are disclosed. The compositions include as an active agent an
```

effective amount of an 8-substituted guanine derivative bonded 9-1' to an aldose having 5 or 6 carbon atoms in the aldose chain. The composition includes a diluent amount of a physiologically tolerable carrier. The guanine derivative is free of electrically charged functionality, while the 8-substituent has an electron withdrawing inductive effect greater than that of hydrogen and contains fewer than about 15 atoms. Animal cellular responses are modulated by contacting the cells with a composition of this invention.

DETD

Con A

CR · cGMP

EAC

FcR

FCS

Guo

DETD

DETD

CLM

L8

ΑN

ΤI

ΙN

PA

PΤ

AΙ

RLI

DT

EXNAM

LREP

CLMN

ECL

DRWN

```
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
                                         bone marrow-derived lymphocytes
                    . . . Cells
              8-bromoguanosine 3',5'-cyclic
8-BrcGMP
             monophosphate
              8-bromoguanosine
8-BrGuo
              concanavalin A
             complement receptor
             quanosine 3',5'-cyclic
             monophosphate
         erythrocyte-antibody complexes
             erythrocyte-antibody-complement
             complexes
             Fc receptor
             fetal calf serum
             quanosine
             8-haloguanosine
8-HGuo
[.sup.3 H]TdR tritium labelled
             deoxyribosylthymidine
Ia antigen
              antigens controlled by the
              immune response genes
IgA, D, E, G. . .
      With reference to Hammett substituent sigma constants for meta
      benzoic acid substituents, the preferred
       8-substituents have positive values. More preferably, the 8-substituents
       have sigma constants of about 0.1 to about 0.7.. .
       . . . interface cells collected separately. Rosettes were lysed in
       0.83% NH.sub.4 Cl, and cells were counted, washed, and used in culture.
       Fc receptor-bearing cells were separated by EA
       rosetting (Parish and Hayward, supra), following the above protocol,
       with the omission of fresh mouse.
      What is claimed is:
         quanine derivative being free of electrically charged functionality,
       and said 8-substituent having a positive Hammett substituent sigma
       constant for meta benzoic acid substituents and
       containing fewer than 15 atoms, together with a diluent amount of a
       physiologically tolerable carrier.
    ANSWER 58 OF 61 USPATFULL
       87:11415 USPATFULL
      Modulation of animal cellular responses with compositions containing
       8-substituted guanine derivatives
       Goodman, Michael G., Carlsbad, CA, United States
       Weigle, William O., Del Mar, CA, United States
       Scripps Clinic and Research Foundation, La Jolla, CA, United States
       (U.S. corporation)
       US 4643992
                               19870217
       US 1983-546679
                               19831101 (6)
       Continuation-in-part of Ser. No. US 1982-439846, filed on 9 Nov 1982,
       now patented, Pat. No. US 4539205
      Utility
       Granted
      Primary Examiner: Hazel, Blondel
       Dressler, Goldsmith, Shore, Sutker & Milnamow Ltd.
      Number of Claims: 4
       Exemplary Claim: 1
       32 Drawing Figure(s); 31 Drawing Page(s)
LN.CNT 2149
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CAS INDEXING IS AVAILABLE FOR THIS PATENT.

LN.CNT 987

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Compositions and methods for their use in modulating animal cellular responses are disclosed. The compositions include as an active agent an effective amount of an 8-substituted guanine derivative bonded 9-1' to an aldose having 5 or 6 carbon atoms in the aldose chain. The composition includes a diluent amount of a physiologically tolerable carrier. The guanine derivative is free of electrically charged functionality, while the 8-substituent has an electron withdrawing inductive effect greater than that of hydrogen and contains fewer than about 15 atoms. Animal cellular responses are modulated by contacting the cells with a composition of this invention.

```
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
DETD
                     . . . Cells
                                           bone marrow-derived lymphocytes
8-BrcGMP
               8-bromoguanosine 3',5'-cyclic
               monophosphate
8-BrGuo
               8-bromoguanosine
               concanavalin A
Con A
              complement receptor
CR
               guanosine 3',5'-cyclic
cGMP
               monophosphate
EΑ
              erythrocyte-antibody complexes
EAC
               erythrocyte-antibody-complement
               complexes
FcR
               Fc receptor
              fetal calf serum
FCS
Guo
               guanosine
8-HGuo
               8-haloguanosine
[.sup.3 H]TdR tritium labelled
               deoxyribosylthymidine
               antigens controlled by the
Ia antigen
               immune response genes
IgA, D, E, G and M
               immunoglobulins.
DETD
       With reference to Hammett substituent sigma constants for meta
       benzoic acid substituents, the preferred
       8-substituents have positive values. More preferably, the 8-substituents
       have sigma constants of about 0.1 to about 0.7.. .
DETD
       . . . interface cells collected separately. Rosettes were lysed in
       0.83% NH.sub.4 Cl, and cells were counted, washed, and used in culture.
       Fc receptor-bearing cells were separated by EA
       rosetting (Parish and Hayward, supra), following the above protocol,
       with the omission of fresh mouse. .
L8
     ANSWER 59 OF 61 USPATFULL
ΑN
       85:56529 USPATFULL
TI
       Conjugate having cytotoxicity and process for the preparation thereof
IN
       Kato, Yoshinori, Hino, Japan
       Umemoto, Naoji, Hino, Japan
       Hara, Takeshi, Hachioji, Japan
       Tsukada, Yutaka, Ebetsu, Japan
       Hirai, Hidematsu, Sapporo, Japan
       Teijin Limited, Osaka, Japan (non-U.S. corporation)
PA.
PΙ
       US 4543211
                               19850924
       US 1983-563858
ΑI
                               19831221 (6)
PRAI
       JP 1982-226237
                           19821224
DT
       Utility
FS
       Granted
EXNAM
       Primary Examiner: Schain, Howard E.
LREP
       Sughrue, Mion, Zinn, Macpeak, and Seas
CLMN
       Number of Claims: 11
ECL
       Exemplary Claim: 1
DRWN
       No Drawings
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A conjugate having cytotoxicity prepared by covalently binding a polymer which has cytotoxic substances linked to its side chains and a reactive

group at its terminal to an immunoglobulin, or its fragment, which is capable of selectively binding to a particular antigen possessed by cells to be killed.

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CAS INDEXING IS AVAILABLE FOR THIS PATENT.
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SUMM . . . the Fc part induces the indiscriminate adsorptive binding to cells other than target cells and also the binding to an Fc receptor on the cell membrane, thus reducing the capability of the conjugate having cytotoxicity to select cells to be killed. Furthermore, . . .

SUMM As concrete examples of the cross-linking agent expressed by formula (XIV), meta-(N-maleimido)benzoic acid ##STR27##

meta-(N-maleimido)benzoic acid 2,4
dinitrophenylester, .beta.-(N-maleimido)propionic acid
N-hydroxysuccinimide ester, etc. may be mentioned.

L8 ANSWER 60 OF 61 USPATFULL

AN 85:52187 USPATFULL

TI Modulation of animal cellular responses with compositions containing 8-substituted quanine derivatives

IN Goodman, Michael G., Carlsbad, CA, United States Weigle, William O., Del Mar, CA, United States

PA Scripps Clinic and Research Foundation, La Jolla, CA, United States

(U.S. corporation)

PI US 4539205 19850903 AI US 1982-439846 19821109 (6)

DT Utility FS Granted

EXNAM Primary Examiner: Hazel, Blondel

LREP Dressler, Goldsmith, Shore, Sutker & Milnamow, Ltd.

CLMN Number of Claims: 20 ECL Exemplary Claim: 1

DRWN 28 Drawing Figure(s); 27 Drawing Page(s)

LN.CNT 1864

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

Compositions and methods for their use in modulating animal cellular responses are disclosed. The compositions include as an active agent an effective amount of an 8-substituted guanine derivative bonded 9-1' to an aldose having 5 or 6 carbon atoms in the aldose chain. The composition includes a diluent amount of a physiologically tolerable carrier. The guanine derivative is free of electrically charged functionality, while the 8-substituent has an electron withdrawing inductive effect greater than that of hydrogen and contains fewer than about 15 atoms. Animal cellular responses are modulated by contacting the cells with a composition of this invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DRWD . . . Cells bone marrow-derived lymphocytes

8-BrcGMP 8-bromoguanosine 3',5'-cyclic

monophosphate
8-BrGuo 8-bromoguanosine
Con A concanavalin A
CR complement receptor

cGMP guanosine 3',5'-cyclic

monophosphate

EAC erythrocyte-antibody complexes erythrocyte-antibody-complement

complexes
Fc receptor

FCS fetal calf serum

Guo quanosine

8-HGuo 8-haloguanosine [.sup.3 H]TdR tritium labelled

deoxyribosylthymidine

Ia antigen antigens controlled by the

immune response genes

IgA, D, E, G.

FcR

With reference to Hammett substituent sigma constants for meta DRWD benzoic acid substituent, the preferred 8-substituents have positive values. More preferably, the 8-substituents have sigma constants of about 0.1 to about 0.7.. . . . interface cells collected separately. Rosettes were lysed in DRWD 0.83% NH.sub.4 Cl, and cells were counted, washed, and used in culture. Fc receptor-bearing cells were separated by EA rosetting (Parish and Hayward, supra), following the above protocol, with the omission of fresh mouse. ANSWER 61 OF 61 USPATFULL L8 85:17723 USPATFULL AN Conjugate having cytotoxicity and process for the preparation thereof ΤI Kato, Yoshinori, Hino, Japan ΙN Umemoto, Naoji, Hino, Japan Saito, Masahiko, Saitama, Japan

Hara, Takeshi, Hachioji, Japan
PA Teijin Limited, Osaka, Japan (non-U.S. corporation)
PI US 4507234

AI US 1983-563860 19831221 (6)

PRAI JP 1982-226236 19821224

DT Utility

FS Granted

EXNAM Primary Examiner: Schain, Howard E. LREP Sughrue, Mion, Zinn, Macpeak and Seas

CLMN Number of Claims: 10 ECL Exemplary Claim: 1 DRWN No Drawings

LN.CNT 1213

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A conjugate having cytotoxicity prepared by covalently binding a serum albumin having cytotoxic substance linked thereto to an immunoglobulin, or its fragment, which is able to bind selectively with a particular antigen of cells to be killed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . it, the Fc part induces the indiscriminate adsorptive binding to cells other than target cells and also the binding to Fc receptor on the cell membrane, thus reducing the capability of the conjugate having cytotoxicity to select cells to be killed. Furthermore, . . .

SUMM As concrete examples of the cross-linking agent expressed by formula (XIV), meta-(N-maleimido)benzoic acid
N-hydroxysuccinimide ester ##STR25## meta-(N-maleimido)benzoic acid 2,4-dinitrophenylester, .beta.-(N-maleimido)propionic acid N-hydroxysuccinimido ester, etc. may be mentioned.